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A CLINICAL AND PATHOLOGICAL RÉSUMÉ OF COMBINED DISEASE OF THE PYRAMIDAL AND EXTRAPYRAMIDAL SYSTEMS, WITH ESPECIAL REFERENCE TO A NEW SYNDROME.

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CONTENTS.

	PAGE
I.—INTRODUCTION	157
II.—DESCRIPTION OF CASE.	
(*a*) Clinical	161
(*b*) Pathological	163
III.—DIFFERENTIATION FROM ALLIED CONDITIONS.	
(*a*) Clinical..	171
(*b*) Pathological	174
IV.—PHYSIO-PATHOLOGY	177
V.—SUMMARY	180
VI.—LITERATURE	180

INTRODUCTION.

A CONSIDERABLE amount of work, both clinical and pathological, has, during the last decade, added to our knowledge of the diseases which affect the striatal system. The conditions which have been investigated have been chiefly those characterized by involuntary movements, or muscular rigidity with akinesia (as seen in paralysis agitans).

Little attention has, however, been paid to cases which clinically show evidence of involvement of both the pyramidal and extrapyramidal tracts. Such cases present signs commonly met with in pseudo-bulbar paralysis on the one hand and paralysis agitans on the other. Brissaud [3] in 1894 recognized the close relationship between these two conditions. Since that time there have been scattered references in the literature

to cases of pseudo-bulbar palsy with signs of paralysis agitans and vice versa.

In 1921, Lhermitte, Cornil and Quesnel [12] described a syndrome under the name "le syndrome de la dégénération pyramido-pallidale progressive," and separated off from the badly defined condition known as pseudo-bulbar palsy a small but distinct group of cases. The joint author of the present paper had an opportunity recently of examining the patient who was the subject of the above communication. The following is a short résumé of the case.

A. B., male, aged 56, enjoyed good health until the spring of 1913, when at the age of 40 he began to experience a pricking and crawling sensation in the left arm and later in the left leg. Accompanying these subjective sensations were a rigidity and weakness which progressed. A year later the limbs on the right side became similarly affected, and about this time difficulties in articulation arose. In 1916 he was no longer able to speak, had considerable dysphagia and commenced to have bouts of involuntary laughing or crying. Within the next two years walking became more and more difficult, so that finally he was bedridden. Since 1917 his condition has remained stationary. At no time was there any apoplectiform seizure.

On examination the face was expressionless, the mouth being held open. The pupils were equal and reacted normally. Ocular movements were full and were quite quickly performed. The facial muscles retained their power to a moderate extent, but movements were carried out more slowly than in a normal person. He could keep his eyes closed against fair resistance and showed his teeth well. The muscles of mastication were hypertonic; their power was fair. The tongue could not be protruded beyond the line of the teeth; it was not atrophied nor did it show fibrillary tremor. The palate moved well on phonation. The patient was unable to speak and could only make a feeble guttural noise. Dysphagia was moderately severe. Involuntary laughing and crying were marked. The head was slightly flexed on the chest. The neck muscles were hypertonic.

Upper limbs.—Both arms were abducted at the shoulder and slightly internally rotated, being held across the body in a flexed position. The forearms were pronated. The hands assumed the posture characteristic of paralysis agitans, namely, flexion at the metacarpo-phalangeal joints to a right angle, with extension at the interphalangeal joints. The tip of the thumb lay in apposition to the terminal phalangeal joint of the index finger.

Tone was much increased in all muscle groups in both arms, though predominating in those determining the posture. No cogwheel phenomenon, however, could be elicited. The hypertonus was greater in the proximal muscles than in the distal. Muscular power was surprisingly good in the upper limbs, but active movements were carried out slowly. There was no muscular atrophy nor tremor. The deep reflexes in the arms were exaggerated.

The muscles of the trunk were hypertonic; the abdominal reflexes were brisk.

Lower limbs.—The lower limbs were powerfully adducted and slightly flexed at the knee-joints. Tone was enormously increased in all muscle groups but especially in the adductors. The power in the legs was comparatively good, but the movements were slow and restricted in range on account of the marked hypertonus. The deep reflexes were brisk and both plantar responses were extensor in type. A flexion withdrawal reflex was easily obtained. Sensation was everywhere normal. Magnus and de Kleijn reflexes were looked for but could not be obtained.

Claude and Alajounanine [4] reported a similar case in 1922 in a man, aged 47, who in 1916 noticed dysarthria and later stiffness in his legs and difficulty in walking. These symptoms progressed gradually. In 1922 he presented the typical picture of pseudo-bulbar palsy. There was complete aphonia with spasmodic laughing and crying. The rigidity of the limbs was excessive, but muscular power was little impaired. He was unable to walk owing to the rigidity of his legs. There were no true bulbar signs such as wasting of the tongue. The deep reflexes were exaggerated. The abdominal reflexes were diminished on the right side. No Babinski's sign could be obtained. The authors considered that this case was similar to that previously reported by Lhermitte, Cornil and Quesnel.

The following are the main points which characterize the pyramido-pallidal syndrome as described by Lhermitte, Cornil and Quesnel.

(1) A gradual onset and slow progression of the disease. There are no sudden vascular accidents such as occur so commonly at the beginning of a case of pseudo-bulbar palsy.

(2) The presence of pseudo-bulbar phenomena such as dysarthria amounting to aphonia, dysphagia and involuntary laughing and crying.

(3) The existence of a marked degree of muscular hypertonus which exceeds considerably in amount that observed in an ordinary case of pseudo-bulbar palsy. This hypertonus combines the characteristics of that observed in both pyramidal and extrapyramidal disease. The rigidity, unlike that seen in an ordinary case of hemiplegia, affects all muscle groups, but predominates in those muscles which determine the posture; further, it is more marked in the proximal muscles than in the distal. The cogwheel phenomenon which is so characteristic of the Parkinsonian rigidity is usually absent.

(4) The posture of the limbs has no distinctive feature except with regard to the hand. In a typical case this assumes the posture of the

hand in paralysis agitans. The wrist is slightly extended, the fingers are sharply flexed at the metacarpo-phalangeal joints and extended (partially or completely) at the remaining joints. The thumb is extended at the interphalangeal joint and its tip is applied to the side of the terminal interphalangeal joint of the index finger. The marked adductor spasm of the lower limbs in Lhermitte's case was very comparable to that observed in long-standing cases of paraplegia from a pure pyramidal lesion. As a rule, however, the posture of the lower limbs has no special characteristics.

(5) The muscular power is surprisingly good, and this fact suggests that the paresis which exists and which is associated with slowness of movement is due less to the pyramidal lesion than to the marked hypertonus, largely extrapyramidal in origin.

In 1921 Ramsay Hunt [6] drew attention to the simultaneous involvement of the pyramidal and extrapyramidal systems in some cases of epidemic encephalitis. He gave the name "pallido-pyramidal type of palsy" to this condition. He found that in these cases the muscular hypertonus combined features peculiar to disturbance of each of these two systems. He noted as especial characteristics of this syndrome the exaggeration of the tendon reflexes, the presence of ankle-clonus and extensor plantar response, in conjunction with a loss of associated movements of striatal type. Out of twenty-five cases of encephalitis showing striatal involvement, only three proved to be examples of this syndrome. He makes no mention, however, of the facies or of the presence or absence of pseudo-bulbar phenomena.

In our experience examples of combined pyramidal and extrapyramidal disease following epidemic encephalitis are uncommon.

One of us [14] has reported a very unusual case showing a unilateral Parkinsonism with a hemiplegia on the opposite side.

In the majority of these cases, however, Parkinsonism is the outstanding feature and the pyramidal tract involvement seems to influence but little the clinical picture of this disease. More rarely pseudo-bulbar symptoms are predominant. One of us has seen such a case, in a woman of 45, in whom there was a clear history of epidemic encephalitis. Thereafter a typical picture of pseudo-bulbar palsy developed, aphonia and laughing and crying being marked. She could not protrude the tongue beyond the line of the teeth; it showed no atrophy. In addition the patient was unable to hold her head up and had marked weakness in her limbs, which were moderately hypertonic. The deep reflexes were increased and both plantar responses were in extension. The Wassermann reaction was negative.

Post-mortem inflammatory lesions were found in the basal ganglia and subthalamic region, with deposits of calcium-iron salts in the anterior part of the globus pallidus and among the cells of the dentate nucleus.

Description of Case.

(*a*) *Clinical.*

The condition which is the subject of the present paper is characterized by signs pointing to pyramidal and pallidal disease with in addition evidence of involvement of the neo-striatum as shown by the presence of choreiform movements.

Job, male, aged 60, enjoyed good health until September, 1915, when he noticed a weakness in his legs, which progressing, necessitated his admission to the Hospital Beaujon, Paris, where he remained a year. In February, 1917, he was transferred to the Hospital Paul Brousse as an incurable.

When examined in September, 1917, by Dr. Levy-Velens, the following were the chief neurological findings: A spastic paraplegia accompanied by a difficulty in using his arms, although they were not paralysed. The deep reflexes in the legs were exaggerated, the right more than the left. Both plantar responses were extensor in type. There were no cerebellar signs or evidence of disturbance of sensibility. The pupils reacted well to light and accommodation. The cerebrospinal fluid showed no increase in albumin nor in the number of cells, and the Wassermann reaction was negative.

From 1917 onwards his condition gradually improved. The motor troubles in his arms and legs lessened and he was able to walk some distance without undue fatigue.

He came under the care of one of us (Lhermitte) in 1920. The following clinical description will illustrate his condition at that time.

General appearance.—He appeared older than his years. There was no lack of expression in his face.

Psychical.—His intellectual faculties were conserved, judgment, memory and reasoning power being normal for his age. There was a tendency to euphoria, but there was no exaggeration of the affective reactions. No involuntary crying or laughing were observed.

Motor power.—Voluntary movements were possible in all four limbs, the head and the trunk. They were full in range but were more slowly performed than by a normal individual. The static muscular force was absolutely normal in all four limbs; the dynamic, on the other hand, was clearly diminished in the upper as well as in the lower limbs. Co-ordination was normal in all four limbs.

Posture.—(1) In the erect posture no troubles of static equilibrium were present. The feet were placed further apart than normally. The legs were slightly flexed at the knee-joints. The muscles of the lower limbs were power-

fully contracted and stood out in relief, especially the quadriceps, glutei and calf muscles.

(2) In the recumbent posture the various muscle groups seemed as strongly contracted as in the erect posture. The left leg was held in a position of flexion at the knee-joint owing to hypertonus in the hamstrings.

(3) In the seated position the hypertonus remained in evidence in the muscles of the trunk and legs ; in other respects the posture was normal.

Gait.—This presented certain striking peculiarities. The patient moved *en masse* in a block-like fashion. The upper limbs were slightly abducted, and the loss of the associated movements of the arms in walking was nearly complete. The lower limbs were advanced somewhat jerkily and this, combined with the excessive muscular hypertonus, caused the patient to take short steps.

Muscular tonus.—No increase in muscular tonus could be detected in the upper limbs. On the other hand, in the lower limbs a marked degree of hypertonus existed both in the recumbent as well as in the erect posture. Active movements diminished the hypertonicity, as also did repeated passive movements. In the recumbent position the patient was able to carry out voluntary movements of flexion and extension quite quickly. It was further noted that a pre-existing condition of hypertonus disappeared when the patient voluntarily activated the opposing muscular group. A position passively imposed upon one of the lower limbs was involuntarily maintained.

Reflexes.—A general condition of hyperexcitability of the deep reflexes was present, but no true ankle or knee clonus could be elicited.

The abdominal and cremasteric reflexes were conserved, but were slightly diminished. The plantar responses were bilaterally extensor in type. A flexion withdrawal reflex (réflexe de défense) could be obtained in each lower limb by appropriate stimulation.

Involuntary movements.—The muscles of the face were the seat of constant involuntary movements, which were typically choreiform in type and consisted of grimaces, blinking of the eyelids and protrusion of the lips, accompanied sometimes by a smacking noise due to the movements of the tongue. They were increased in intensity and range by the act of walking, or when the patient used his arms. At times, especially when he was fatigued, movements of a similar character appeared in the left hand.

Synkinesiæ and associated movements.—These phenomena were well developed. The following tests were employed :—

(1) On extending or flexing the lower limb, the patient being instructed to resist the movement, extension or flexion followed involuntarily in the other lower limb.

(2) Passive movements of flexion of the lower limbs at the knee-joint resulted in involuntary extension of the toes and particularly of the hallux.

(3) On asking the patient to grip an object firmly in one hand, an involuntary contraction in certain muscle groups in the opposite arm occurred.

(4) With the patient in the upright posture, a passive movement of exten-

sion of the head and trunk resulted in a spontaneous bilateral extension of the great toes.

Sensation.—All forms of sensation were normal. There were no subjective sensory symptoms.

Vasomotor system, normal. *Sphincters*, normal.

Eyes.—No paralysis of the intrinsic or extrinsic ocular muscles. The pupils reacted normally to light and accommodation. The fundi were normal No hemianopia was present. Vision was normal.

Examination of the ears and throat.—Hearing was normal. Labyrinthine reactions (Bárány) were normal. Taste was unaffected. Olfactory sense was slightly diminished. The muscles of the pharynx and palate contracted normally. The uvula and palate presented involuntary movements of a choreiform nature. Voluntary power in the vocal cords was normal, but at rest they were the seat of involuntary movements of abduction and adduction. Speech was dysarthric.

Other findings.—Heart and lungs were normal. The arterial tension was raised. Wassermann reaction in the blood was negative.

Résumé of Clinical History and Findings.

A male, aged 60, first noticed weakness in his lower limbs at the age of 52, which progressed, and in a comparatively short time rendered walking a matter of considerable difficulty. At that time he showed a spastic paresis with evidence of a bilateral pyramidal lesion. There was also considerable motor difficulty in the upper limbs, but no true paralysis could be detected in any of the limbs. At the age of 56 he began to improve, and finally could walk a considerable distance without fatigue. In 1921, at the age of 58, he presented the clinical picture of a paralysis agitans *sine tremore*, and without involvement of the facial muscles. A bilateral extensor plantar response was obtained as previously. Involuntary movements, choreiform in type, were present in the muscles of the face, pharynx, larynx, and at times in those of the left hand. There was also a considerable articulatory difficulty.

Death took place in 1923.

(*b*) *Pathological Findings.*

Methods.—The brain and spinal cord, after hardening in formalin, were dealt with in the following manner: The two hemispheres of the brain were separated by a median longitudinal section through the corpus callosum, in the usual manner. One hemisphere was then cut coronally into convenient slabs. These, after preliminary treatment by the method of Weigert-Pal, were cut in whole sections by means of the large microtome of Nageotte. The block containing the basal ganglia was cut in series, every tenth section being taken.

Large serial sections of the cerebellum and pons were also treated by the method of Weigert-Pal, as were sections of the medulla and cord. The basal ganglia of the other hemisphere were cut longitudinally and stained by a variety of methods. Pieces of cortex, mid-brain, medulla and cord were similarly treated.

(1) *Cortex.*—Weigert-Pal sections showed a normal staining of the cortical fibres. By Nissl's method, the cortical cells appeared normal; there was slight neuroglial cell proliferation. The vessels showed an *état précriblé* and *criblé*.

FIG. 1.—*Case* Job ——. Coronal section through the left hemisphere showing a reduction in volume of the caudate and lenticular nuclei. The ansa lenticularis is thinned. The internal capsule, optic thalamus, and capsule of the corpus Luysii appear normal. Method of Weigert-Pal. (Natural size. Drawing.)

(2) *Basal ganglia. Macroscopically.*—When a large coronal Weigert-Pal section was looked at with the naked eye or by means of a hand lens, and compared with normal controls, the corpus striatum appeared smaller than normal. The caudate nucleus, especially its body, was diminished in volume. The ansa lenticularis was definitely diminished in breadth.

The external capsule appeared paler than normal. The markings in the putamen, indicating the fibre bundles, were much fewer in number when compared with those present in a normal control.

The lateral ventricles were moderately dilated.

Microscopically :—

(1) *Caudate nucleus.*—There was a diminution in the number of fibres running from the caudate to the putamen. The cells, however, did not show a diminution in number, and appeared for the most part normal. There was no neuroglial cell overgrowth.

(2) *Putamen.*—We have previously remarked that the usual markings indicating bundle fibres in the putamen were considerably less than in the normal, and this isborne out by microscopical examination of Weigert-Pal sections (both coronal and longitudinal).

The bundles, though not obviously diminished in number, showed in the majority of the sections a pronounced pallor owing to degeneration of the fibres composing them. In some bundles only two or three fibres possessed

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FIG. 2. FIG. 3.

FIG. 2.—Putamen from a normal control. Method of Weigert-Pal. × 50.

FIG. 3.—Putamen. *Case* Job ——. The fibre-bundles appear pale and are reduced in number. Method of Weigert-Pal. × 50.

a normally staining myelin sheath. These changes were particularly striking in the bundles of fibres entering the globus pallidus.

The external medullary lamina was thinned and contained many degenerated fibres. (These are fibres coming from the caudate nucleus and putamen which run down in the external medullary lamina to join the ansa lenticularis). Some fibres of the external capsule were also degenerated.

Sections of the putamen stained by Nissl's method showed considerable changes in the cellular structures of this region. The polygonal cells of the putamen were considerably reduced in number. In order to arrive at a more accurate estimation of the number of cells, a method of counting was adopted. Using a one-sixth objective and a No. 4 eye-piece, the cells in six different fields were counted and an average arrived at. A section of the putamen at the same level in a normal control of about the same age was treated in the

same manner. By comparing the counts it was found that the cells of the putamen were reduced by approximately one-half.

The remainder of the cells showed in general pathological changes, such as

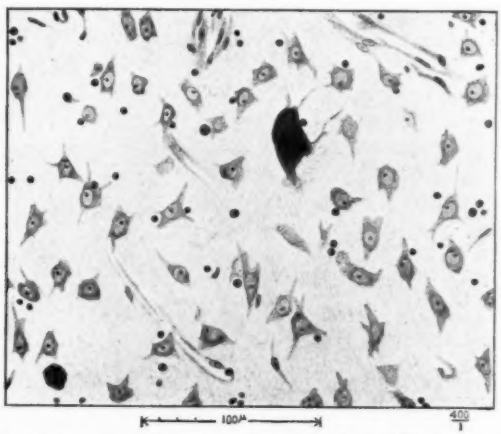

FIG. 4.—Putamen. Normal control. Method of Nissl. × 400.

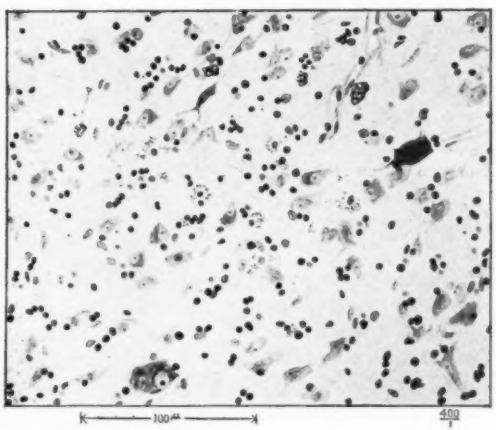

FIG. 5.—Putamen. *Case* Job ——. The small polygonal cells show chromatolytic changes. There is considerable neuroglial cell overgrowth. Method of Nissl. × 400.

pallor, swelling of the cell body, eccentricity of nucleus, satellitosis, &c. The Golgi I type of cell in the putamen were less affected, but some showed varying degrees of neuronophagia.

In sections stained by hæmatoxylin and eosin and by the method of Nissl,

there was observed a considerable overgrowth of neuroglial cells. These cells contain oval or rounded nuclei with a pale protoplasm, showing a number of chromatin points. Certain of these neuroglial cells, especially those in relation

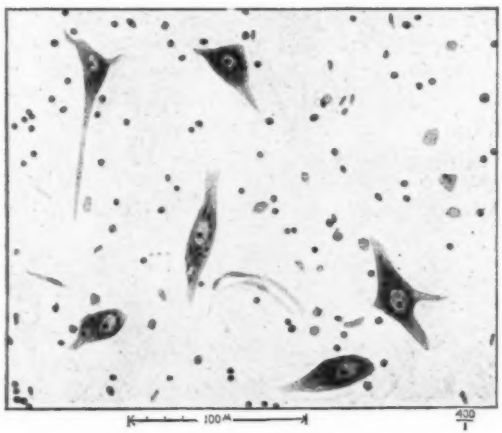

FIG. 6.—Globus pallidus. Normal control. Method of Nissl. × 400.

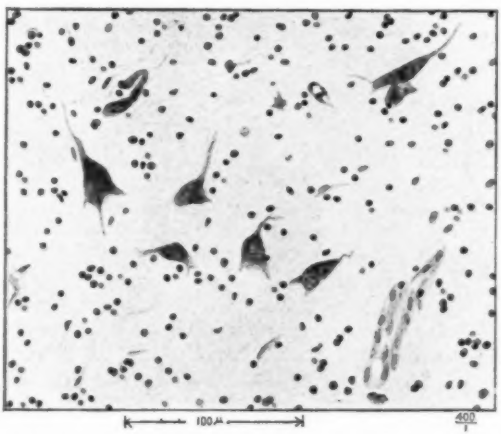

FIG. 7.—Globus pallidus. *Case* Job ——. Some of the cells appear shrunken and contain an abundance of lipochrome pigments. Their dendrites are reduced in volume. Method of Nissl. × 400.

to vessels, contained a quantity of lipochrome pigment. Rod-shaped cells were numerous.

These changes, namely, reduction in the number of cells in the putamen

with accompanying neuroglial cell overgrowth, were observed at all levels and in all parts of the putamen. As the subthalamic region was approached the increase in the neuroglial element became more marked, especially in the posterior half of the putamen. The vessels in the putamen showed well-marked *état criblé*. The walls were thickened and showed an increase of the cellular element. The neuroglial cell overgrowth was particularly intense in relation to the vessels. Hæmorrhages into the perivascular spaces were not confined to vessels in this region, but were more marked here than elsewhere.

Globus pallidus.—In the outer part of the globus pallidus many of the fibres arising in the putamen failed to stain. On the other hand, the pallido-thalamic fibres running transversely (coronal sections) to enter the thalamus stained normally. In horizontal sections transversely cut bundles of fibres

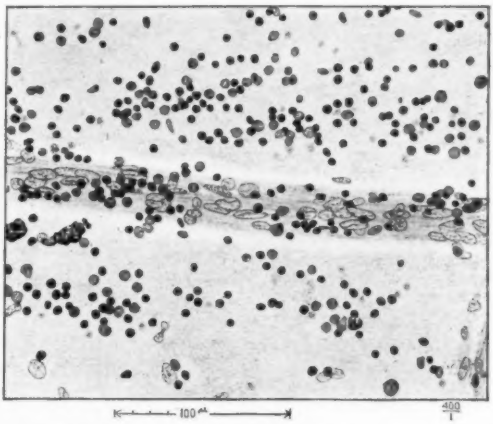

FIG. 8.—Putamen. *Case* Job ——. Marked proliferation of the neuroglial element in the region of a vessel. The cells of the putamen appear reduced in number. Method of Nissl. × 400.

were seen, many of which showed definite alterations in their myelin sheath. These were fibres arising from the motor cells of the globus pallidus which then enter the ansa lenticularis.

The cells of the globus pallidus showed certain changes both in their number and in their form. Using the counting method in a similar manner as that employed in enumerating the cells of the putamen, but with a low power (two-thirds instead of one-sixth), the average count per field was thirty-six as compared with forty-four cells per field in the normal control. This reduction of cells was relatively small, and was much less than that found in six cases of Parkinson's disease, in which the reduction in cells was never less than one-half the normal count. Many of the cells were normal, but some showed varying degrees of degeneration.

Neuroglial cell overgrowth in the globus pallidus was relatively slight and the contrast with the putamen, in this respect, was striking.

The vessels, as elsewhere, showed an *état précriblé* and *criblé*, but rarely was there an appearance of proliferation of the cellular element in the walls of the vessels, as was found in the putamen. No deposits of iron, either in

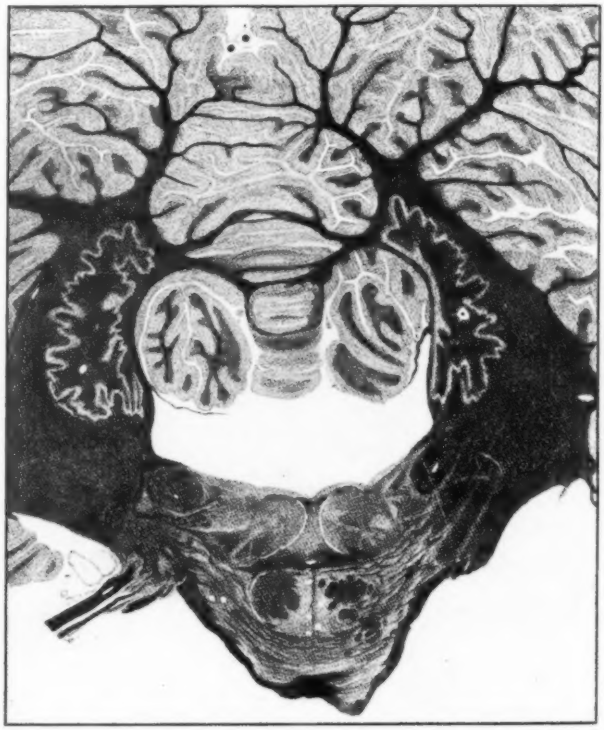

FIG. 9.—Transverse section through the lower part of the pons. The cerebellar cortex, dentate nuclei, tegmentum and pyramidal fibres appear normal. Method of Weigert-Pal. Natural size.

the form of globules or in the walls of vessels, were present in the globus pallidus.

Thalamus.—The cells appeared normal. The pallido-thalamic fibres were approximately normal. There was little if any neuroglial cell overgrowth.

Subthalamic region.—As previously mentioned, the changes in the putamen were as marked in this region as at a higher level; their greatest intensity was in the posterior part of the putamen (horizontal section).

Substantia innominata.—In horizontal sections some of the cells in the posterior part of the nucleus showed degenerative changes, but more anteriorly they appeared normal. The neuroglial cell overgrowth was slight and contrasted with the marked increase of these cells in the adjacent part of the putamen. A vast number of amyloid bodies were present in this region, but this is not an uncommon finding in this location: it was relatively more marked posteriorly in the area in which some of the nerve cells showed pathological alterations.

Corpus Luys.—The volume of this appeared normal, as did the cells composing it. No increase in neuroglial cells was present. The fibres composing its capsule were normal.

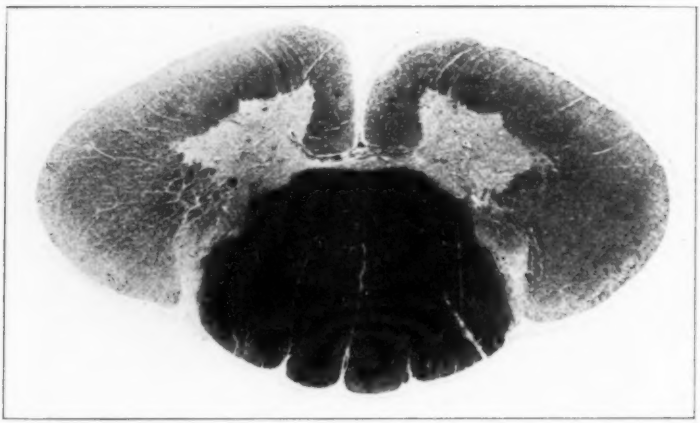

FIG. 10.—Section through the cervical region of the cord, showing the degeneration of the crossed pyramidal tracts, more evident on the left side. (The marginal pallor is commonly met with in persons over the age of 50 and is of no importance.) Weigert-Pal. Photomicrograph. × 20.

Forel's bundles H_1 and H_2.—Normal in appearance.

Field of Forel.—Normal in appearance.

Zona incerta.—Normal.

Ansa lenticularis.—As previously mentioned, this was diminished in volume, owing to the degeneration of many of the fibres from the putamen and some from the globus pallidus.

Internal capsule.—No degeneration of the motor pyramidal fibres in the interior capsule was observed.

Red nucleus.—At all levels the cells composing the nucleus were normal.

Bundle of Vicq d'Azyr.—In Weigert-Pal sections this appeared normal.

Mid-brain.—The substantia nigra was normal. There was no degeneration of the fibres in the pes pedunculi.

Medulla.—At this level a slight but definite degeneration of the pyramidal fibres was observed; this degeneration could not be traced into the pons.

Spinal cord.—Beyond a moderate but quite evident bilateral degeneration in the lateral column in the region of the crossed pyramidal tract, the fibre tracts in the cord appeared normal.

Résumé of Pathological Findings.

The following were the main features of the pathological findings:—

(1) *Putamen.*—A marked reduction in the cells of the putamen, with accompanying neuroglial overgrowth, which, although not absolutely confined to this region, was more marked here than elsewhere. A degeneration of many of the fibres running from the putamen into the globus pallidus and ansa lenticularis; many of the fibres having origin in the caudate nucleus were also affected.

(2) *Globus pallidus.*—A slight but definite reduction in the number of the motor cells, neuroglial cell overgrowth which was less than in putamen, and a degeneration of certain of the fibres arising in the globus pallidus, especially those which go to compose the ansa lenticularis.

(3) Degeneration of the pyramidal tract not evident above the level of the medulla. In the cord degeneration of the crossed pyramidal tracts.

Differentiation from Allied Conditions.

(*a*) *Clinical.*

The following are the chief neurological points in the case:—

(1) The presence of a pyramidal lesion is evidenced by the increased deep reflexes and the bilateral extensor plantar responses. That the lesion in the pyramidal tracts was not severe or widespread is suggested by the absence of true paralytic phenomena.

(2) The clinical symptoms further indicate that the function of the extrapyramidal system was interfered with. The Parkinsonian rigidity, slowness of movement and loss of associated movements, point to a lesion of the pallidal system, while the choreiform movements of the face, larynx and pharynx are indicative of a lesion either of the neostriatum (putamen or caudate) or of certain structures in the subthalamic region.

Clinically the present syndrome may be differentiated from the following conditions:—

(1) *Parkinson's disease.*—The presence of a pyramidal lesion and of choreiform movements at once distinguishes it from this disease. It

has, however, certain features in common with this affection, namely, muscular hypertonus, akinesia, &c., which are identical with those found in paralysis agitans.

(2) *Pseudo-bulbar palsy.*—It may be distinguished from the pure type of this disease by the absence of dysphagia, spasmodic laughing and crying and the presence of a marked degree of muscular hypertonus, extrapyramidal in type, with in addition choreiform movements. Articulatory difficulty and a double pyramidal lesion are common to the two affections.

(3) *Progressive pyramido-pallidal degeneration.*—The principal points of this syndrome have been discussed earlier in this paper. In the present case choreiform movements are added to the typical picture of this condition.

Spastic pseudo-sclerosis.—Creutzfeldt [5], and later Jacob [7], have described a syndrome, clinical and pathological, which in certain respects bears comparion with our case. Under the term "spastic pseudo-sclerosis" these authors have described a condition which affects the entire central nervous system and which resembles the pseudo-sclerosis of Strumpell-Westphal.

The affection appears about middle age or later; its etiology remains unknown. The initial symptoms are mental and consist of periods of excitation or depression. Later pain and paræsthesiæ are experienced in the lower limbs, which become somewhat rigid. At this early stage the only positive sign is an abolition of the abdominal reflexes. At a later stage the gait is ataxic, difficult, and frequent falls occur; finally walking becomes impossible. Amongst other important signs are a lack of spontaneous movements, mobile spasms, tremor and athetotic movements and dysarthria. The deep reflexes are active, less often diminished or even absent; the sign of Babinski is positive on both sides. The mentality of the patient now shows further retrograde changes, which consist of a marked apathy and negativism. Sometimes delirium or a condition of confusion with hallucinations make their appearance. The ocular fundi show no change, and the blood and cerebrospinal fluid are normal.

The evolution of this condition is comparatively rapid and it is usual for the disease to run its course to a fatal termination within a year.

The complexity of the symptomatology makes it easy for this condition to be confused with others, such as disseminated sclerosis and general paralysis of the insane.

Pathologically this syndrome is characterized by the wide dissemination of the lesions which are unrelated to vessels and which affect principally the frontal and temporal lobes, the corpus striatum, the ventro-median nuclei of the thalamus, the bulbar nuclei and the anterior cornual cells in the cord. In the cortex the Betz cells and the third and sixth layers are principally affected. The striatum shows a disappearance of many of the small polygonal cells. The locus niger is less affected. As a result of the lesion of the motor cells in the precentral gyrus there is a slight degeneration of the pyramidal tracts in the cord.

Undoubtedly the syndrome of Creutzfeldt and Jacob possesses features both clinical and pathological which lend it considerable interest, but it is questionable whether one can accept it as a definite clinical entity.

Although we do not wish to discuss in detail the pathogenesis of this condition, as described by Creutzfeldt and Jacob, the resemblance between the pathological changes and those occurring in epidemic encephalitis seems a close one. We know that epidemic encephalitis is possessed of a pathological basis, extremely varied in its character, consisting in degenerative lesions, and at other times vascular lesions, typical of an acute inflammatory condition. The latter may be very discrete or absent, while the former change may be very marked, and may even constitute the entire pathological picture. Further, in one of Jacob's cases of spastic pseudo-sclerosis, in addition to degenerative lesions there were discrete but indisputable inflammatory changes around vessels of the pia mater, the pathological picture resembling closely that of encephalitis.

Apart from this aspect of the syndrome of Creutzfeldt and Jacob, this condition is quite different, both from a clinical and pathological standpoint, from the case which we have described in the present paper. Clinically by the absence of mental changes, the presence of choreiform movements and the slowness of the evolution of our case, and pathologically by the incidence of the disease on the strio-pallidal and the pyramidal systems. Contrary to the finding of Creutzfeldt and Jacob, the degeneration of the cortico-spinal tracts is not in our case the result of a cortical lesion, as the former could not be traced higher than the medulla, and no degeneration of the motor cortical cells was present, the pyramidal tracts being affected directly by the unknown pathogenic agent which produced the disease.

Amyotrophic lateral sclerosis.—The form of this disease, which

spreads upwards to involve the bulbar nuclei, may give a clinical picture which at first glance might resemble the present syndrome. The difficulty in speech, the expressionless aspect of the lower face, the muscular hypertonus, especially of the lower limbs, with evidence of a double pyramidal lesion, are signs which might deceive. However, in the present case, the absence of atrophy of the tongue and wasting of the upper limbs and the presence of muscular rigidity with the conservation of muscular power, and, in addition, choreiform movements, are points that easily separate the two conditions.

(*b*) *Pathological.*

(1) *Paralysis agitans.*—It is now recognized that from the pathological standpoint there are two forms of this disease : (*a*) presenile ; (*b*) senile. The former occurs at an earlier age than the latter (e.g., between the ages of 40 and 60). The morbid changes consist of a primary degeneration of the large cells of the globus pallidus, the number of which is greatly reduced. This change is accompanied by a proliferation of the neuroglial cells. This cellular affection is akin to that found in the cord in cases of amyotrophic lateral sclerosis. These changes are, however, not confined to the globus pallidus, but, as the Vogts [18], Lhermitte [11], Bielchowsky [2] and Lewy [10] have shown, the small polygonal cells of the putamen and caudate nucleus are also affected, though, as a rule, to a less extent than those of the globus pallidus. Further, the Vogts have pointed out that in those cases characterized clinically by tremor the cellular affection is more intense in the striatum than in the pallidum, while the cases in which rigidity is marked and tremor at a minimum or absent show more striking changes in the pallidum than in the putamen or caudate nucleus.

(*b*) In the *senile* type the most striking changes are in the neighbourhood of the vessels. Perivascular disintegration with cavity formation (lacunæ) leads to a secondary destruction of the nerve cells.

In a certain proportion of cases of paralysis agitans changes in the substantia nigra are present. Lewy [10] in a series of fifty-two cases only found such changes in 14 per cent.

Trétiakoff [17], on the other hand, considers that such changes are primary, but we think that it is more likely that, when present, they are secondary to the destruction of cells in the globus pallidus. They are similar to the changes found in the anterior horn cells in cases of hemiplegia, or in the cells of the inferior olive, following primary degeneration of the cerebellar cortex.

(2) *Parkinsonian syndrome following epidemic encephalitis.*—A large amount of pathological data has now accumulated in connection with the syndrome. The changes, though often widespread, have their greatest incidence in the mid-brain, especially on the cells of the substantia nigra. These changes, unlike those in the idiopathic form of paralysis agitans which are often limited to a portion of one or both substantia nigra, are more or less uniform throughout both substantia nigra, and consist in a destruction of the pigmented cells with liberation of free pigment. Neuroglial cell overgrowth is as a rule prominent. The vessels show various changes indicating an existing or previous inflammatory process. In nine cases investigated by one of us (McAlpine) the cells of the substantia nigra were markedly affected, while those of the globus pallidus showed little or no reduction in numbers, and were for the most part healthy.

(3) *Syphilitic encephalitis of the corpus striatum*—Little attention has been paid to this condition, especially from the pathological standpoint, judging by the paucity of the references in the literature.

The majority of such cases show signs of Parkinsonism, whilst a few present the typical picture of pseudobulbar palsy. An Argyll Robertson pupil is nearly constant.

Lhermitte [11], in four cases of syphilitic Parkinsonism, found a striking reduction in the number of the cells of the globus pallidus, and sometimes a degeneration of the pallidal fibres. The substantia nigra was only slightly involved. That which gives a distinctive appearance to the pathological picture are the changes in the vessel walls, which consist of a perivascular infiltration with plasma cells and lymphocytes. In addition a chronic syphilitic meningitis is usually present. The morbid process, though not confined to the corpus striatum, produces cellular changes only in this part, and to a much less extent in the substantia nigra. Wilson and Cobb [20], in a paper entitled "Mesencephalitis Syphilitica," have collected from the literature a number of cases of paralysis agitans accompanied by either tabes dorsalis or general paralysis of the insane. They also give a clinical account of four similar cases personally observed. They conclude that "an Argyll Robertson pupil and tremor occurring in the same case of syphilis are not merely coincidental, as held by most former investigators of the subject. Rather does this combination (with its various elaborations, such as oculomotor palsy and rigidity) indicate an incidence of the syphilitic process on the mid-brain, a true syphilitic mesencephalitis."

They base their views on the generally accepted theory as to the mid-brain localization of the Argyll Robertson pupil, and on the alterations in the substantia nigra described by Trétianoff [17] in cases of idiopathic paralysis agitans.

In this connection it is noteworthy that three out of the four cases of Lhermitte's referred to above showed an Argyll Robertson pupil and that the maximum lesion lay in the corpus striatum. Wilson and Cobb refer to a similar case, published by Mellar and Katz [15], which showed a marked syphilitic inflammation of the corpus striatum with an outfall of the cells of the globus pallidus.

C. and O. Vogt [18] describe a case of Westphal's' "P. Grohe," which showed paralysis agitans with double Argyll Robertson pupils. Pathologically a lacunar condition existed in the striatum pallidum to which the Vogts attribute the paralysis agitans.

Pappenheim [16] refers in a recent paper to similar cases published by Petter and Jacob, and describes a case of his own which is of considerable interest. This concerned a male, aged 41, who had a definite attack of epidemic encephalitis in December, 1921. Thereafter he developed typical Parkinsonism. In May, 1923, the pupils reacted normally, but the left ankle-jerk was diminished and the right one could not be obtained. He complained at times of sharp pains in his right leg. The cerebrospinal fluid examined on several occasions showed a positive Wassermann reaction, 60 cells per cubic millimetre, a marked increase in the amount of globulin and a curve with colloidal gold suggesting meningo-vascular syphilis. The patient died in March, 1924. On microscopical examination of the brain marked inflammatory changes involving the meninges and parenchyma were apparent. The majority of the cells of the globus pallidus were intact. The substantia nigra, on the other hand, was markedly altered. The few cells that remained showed chromatolysis. No noteworthy changes, apart from those of the vessels and meninges, were found in other parts of the brain. In the lower part of the cord, in addition to a meningitis, there was involvement of the root entry zone with a degeneration of some of the fibres in this region, and to a less extent in the posterior columns.

Pappenheim concludes that in this case the brain showed evidence of a syphilitic infection as shown by the meningitis and cord changes, but that the Parkinsonism was post-encephalitic in view of the marked alteration in the substantia nigra.

The mere presence of a fixed pupil or of an Argyll Robertson pupil

in a case of Parkinsonism does not necessarily indicate that syphilis is the causal factor, as these pupillary changes may be the permanent relic of an attack of epidemic encephalitis.

With regard to Trétiakoff's hypothesis of the mid-brain origin of paralysis agitans to which Wilson and Cobb refer, it should be noted that his results were based on the examination of the mid-brain alone in cases of paralysis agitans; in none of his cases were the basal ganglia examined. Therefore it seems to us that there is no pathological evidence in support of the view of Wilson and Cobb that the rigidity and tremor in cases of syphilitic Parkinsonism associated with an Argyll Robertson pupil, and other signs of syphilis, are due to a mesencephalic lesion alone. It should be added, however, that these authors at the commencement of their paper write: ". . . . hence the lesions causing the syndrome under discussion may be more widespread than the title of this paper would indicate."

Physio-Pathology.

The pathological findings have shown us that the morbid process in the case described in this paper was not confined to one system, extrapyramidal or pyramidal, but involved both. The pyramidal lesion, though not intense, was clearly seen in the degeneration of the crossed pyramidal tracts, and clinically by a double extensor response. That the degeneration of these tracts was only partial was shown clinically by the absence of paralytic phenomena in the lower limbs.

The lesion of the striate body (globus pallidus, caudate and putamen) was not confined to one portion but involved both the neo- and the paleo-striatum. The pathological changes were most marked in the putamen, less in the outer segment of the globus pallidus and were scarcely present in the inner segment. In other words, the intensity of the morbid lesion diminished progressively from without inwards. To the lesions in the putamen and in the caudate nucleus can be attributed the choreiform movements in the facial, laryngeal and pharyngeal muscles. The work of Jelgersma [8], Alzheimer [1], Kleist [9], P. Marie and Lhermitte [13], and the Vogts [18], have shown that in Huntingdon's chorea, in the caudate nucleus and putamen the greatest incidence of the disease may be found, the globus pallidus being usually intact. These authors conclude that the involuntary movements may be attributed to a lesion of the striatal cells. A difficulty, however, arises, from the fact that according to the Vogts the tremor in paralysis agitans is due to a lesion in the putamen. Those cases which clinically

showed tremor were found to have lesions of greatest intensity in the putamen and less in the globus pallidus. The opposite findings were observed in those cases in which the tremor was slight or absent and in whom rigidity was marked. The interesting point in the present case is the apparent substitution of choreiform movements for tremor.

Pathologically we have shown that the globus pallidus presented definite changes, though these were less than those in the putamen. Such changes would account for the Parkinsonian rigidity, the loss of associated movements, &c., which were present.

Lastly, mention may be made of the influence of a lesion of the pyramidal tract on choreiform movements. It is well recognized that a lesion of the internal capsule, such as a thrombosis or hæmorrhage, occurring in a person previously affected with chorea, arrests involuntary movements, while the complete destruction of the motor cortex alone stops for a time only the movements of chorea or athetosis or the tremors of paralysis agitans. But in our case the pyramidal lesion has not completely abolished the involuntary movements in the limbs but has markedly reduced it. Moreover, it is interesting to note that it is precisely in the musculature of the face, tongue and pharynx that the involuntary movements are most marked, and that cortico-ponto-bulbar fibres from these parts showed little or no degenerative changes. As one of us has previously pointed out, the essential condition for the development of choreiform movements is a diminution in muscular tonus (Lhermitte), which is clearly seen in Sydenham's chorea as well as in the variety described by Huntingdon. Should for any reason a hypertonus, pyramidal or extrapyramidal, be grafted on to a case of chorea, the involuntary movements abate considerably and even disappear. It would appear then that in this pyramido-striatal syndrome which we have described the choreiform element has been reduced in extent as a result of the supervention of a hypertonus, both extrapyramidal and pyramidal in nature.

Kinnier Wilson [19] in his recent Croonian Lecture discusses, *inter alia*, the pathogenesis of chorea and athetosis. He emphasizes the fact that the presence of involuntary movements, as seen in these two conditions, presupposes the relative integrity of the cortico-spinal tracts, and cites several cases in which destruction of these tracts led to the disappearance of a pre-existing choreo-athetosis. He mentions the case of a girl, aged 14, suffering from an infantile cerebral hemiplegia with athetosis and Jacksonian epilepsy since the age of 1 year, in whom the athetosis disappeared for about twenty minutes following

each fit. He attributes this phenomenon to a temporary paresis of the cortico-spinal system. He then adds without further explanation, "The evidence in this case pointed unmistakably to a cortical origin for all the symptoms." He had referred to this case, however, in a previous publication in the following words:—

"This paralysis was of cortical origin. Clearly, were the impulses producing the athetoid movements reaching the spinal centres by some other route, the latter would still be open to their influence and the movements should continue." We are in total agreement with his explanation of the temporary cessation of the movements, i.e., as being due to a paresis of the cortico-spinal tract, but we are unable to understand from the above statements on what grounds he postulates a cortical origin for the athetosis in this and similar cases.

Wilson believes that "the choreo-athetotic activity is the activity of cortical reflex arcs; the movements have cortical quality and yet they are involuntary." He presupposes a higher motor centre in front of the Rolandic area. Afferent impulses reaching this centre by means of the cerebello-mesencephalo-thalamo-cortical fibres are interfered with and the lower motor centre (Betz cells) is no longer inhibited by the higher centre. In support of the part played by the cerebello-mesencephalo-thalamo-cortical tract, he mentions on the clinical side atonia and the tendency to a pendular knee-jerk met with in cases of chorea, and anatomically the occurrence of chorea in lesions of the superior cerebellar peduncle.

No one denies the existence of thalamo-cortical fibres to the frontal region, but it seems to us a far step into the realms of speculation to assign to these fibres functions such as Wilson has outlined.

It would seem best in tackling a problem of this kind to start with a disease exhibiting involuntary movements, in which the pathological basis has been fairly well established. Huntingdon's chorea would seem to meet the description tolerably well. The principal morbid changes described by most investigators into this disease have been found in two situations: (1) a widespread cellular atrophy and demyelinization in the frontal cortex; to these changes are attributed the mental alterations so characteristic of the advanced stage; (2) a disappearance of many of the cells of the caudate nucleus and putamen, with neuroglial cell overgrowth. It is generally held that the choreiform movements are due to these changes in the neostriatum.

Wilson in his Croonian Lecture makes no allusion to Huntingdon's chorea and other closely allied conditions associated with pathological

changes in this region. In a previous communication, however, he states: "We must, of course, accept the occasional occurrence of choreo-athetotic movements from lesions of the corpus striatum."

We believe that by virtue of the evidence afforded by the present case and the anatomo-pathological basis of Huntingdon's chorea, no theory as to the pathogenesis of choreo-athetosis is adequate that does not take into account the neostriatum as a factor in the production of such movements.

Summary.

(1) A brief résumé is given of conditions in which signs of both pyramidal and extrapyramidal disease occur.

(2) A case is described in which clinically there appeared rigidity with akinesia, choreiform movements and signs indicating a pyramidal tract lesion, and pathologically alterations in the corpus striatum, especially in the putamen, with slight degeneration of both pyramidal tracts.

(3) This syndrome is differentiated both clinically and pathologically from other conditions resembling it.

(4) The physio-pathological basis of chorea is briefly discussed.

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LYMPHORRHAGES IN THE MUSCLES IN EXOPHTHALMIC GOITRE.

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AND

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In this communication the results of the examination of the viscera and muscles in nine cases of exophthalmic goitre are recorded.

The extrinsic eye muscles and the heart muscle have been examined in every case, and of the skeletal muscles the deltoid, rectus and biceps were examined.

The first case noted by one of us (L. S. D.) was in 1911, where on examination of the muscles of a fatal case of exophthalmic goitre, lymphorrhages were found, most markedly in the biceps. During 1924-25, a further eight cases have been examined, in seven of which well marked lymphorrhages have been present in the muscles. One of these seven cases was suffering from a combination of exophthalmic goitre and myasthenia gravis, which adds considerably to the interest of the findings.

Examination of the Muscles.

The muscular tissue was fixed in Kaiserling's fluid and embedded in paraffin. Sections were cut in longitudinal and transverse section of the fibres. The longitudinal sections appeared to offer a more satisfactory picture of the size of the lymphorrhages and of their relative position among the muscle-fibres. The sections were stained with hæmalum and eosin, and with van Gieson's stain, but the cell contents of the lymphorrhages were most clearly defined in sections stained with Giemsa stain which afforded beautiful pictures.

The lymphorrhages varied considerably in size from a few cells aggregated together between the muscle-fibres, to a wide tract of mononucleated cells extending for a considerable distance among the fibres and producing a wide separation of them. In some of the collections of cells both small and large lymphocytes were visible. The actual size and number of lymphorrhages were far greater in some cases

than in others. In every case the eye muscles were more severely affected than the skeletal muscles, and the cardiac muscles to the least extent.

The cell content of the lymphorrhages, as already stated, was most clearly defined in the preparations stained with Giemsa stain. The majority of the cells were lymphocytes, amongst which were scattered a few endothelial and plasma cells. In several instances the lymphorrhages were found to be in close proximity to small blood-vessels. In addition to the lymphorrhages a proliferation of interstitial cells was frequently observed, and not infrequently a chronic interstitial myositis

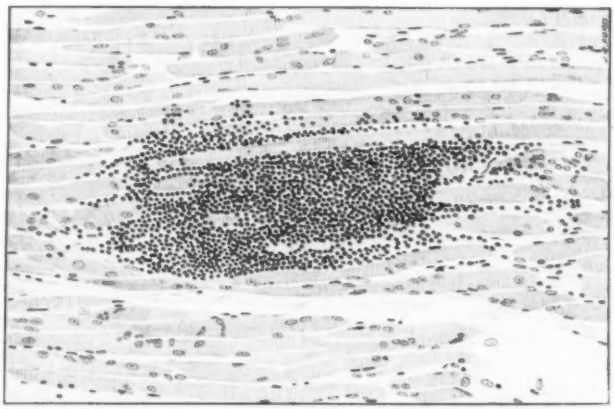

FIG. 1.—*Case* 8. Graves's disease. Longitudinal section of an eye-muscle showing a "lymphorrhage." Magnification: Zeiss D, ocular B.

with atrophy of the muscle-fibres to varying degrees was a pronounced feature in the affected tissues. Similar muscular atrophy was also present in proximity to the larger lymphorrhages.

Experience has shown us that it may be necessary to examine several muscles and many sections of the individual muscle before lymphorrhages are found.

THYMUS AND THYROID GLANDS.

The thymus gland in three cases was large and showed the usual microscopical appearances met with in this gland in exophthalmic goitre. There were no changes present which could be regarded as abnormal.

Thyroid.

In every instance of the series the thyroid gland presented on naked-eye and microscopical examination the usual appearances met with in exophthalmic goitre. In four out of the nine cases the lymphoid tissue in the thyroid gland was a marked feature.

TABLE I.

Case Number	Sex	Age	Duration of symptoms	Exophthalmos	Treatment	Muscle lymphorrhages
1	F.	32	Few years	Nil	Surgical	Marked
2	F.	39	Three years	Present	Iodine and X-rays	Present
3	M.	22	Three years	Marked	Surgical	Slight
4	M.	48	Few years associated with myasthenia gravis	Nil	Thyroid and pituitary and insulin	,,
5	F.	26	Four and a half years	Present	Radium	,,
6	F.	57	Nine years	Marked	Surgical	Marked
7	M.	53	A few years	Slight	,,	None
8	F.	33	,,	Marked	,,	Marked
9	F.	35	Three years	,,	Medical	,,

Literature.

Weigert in 1901 recorded a case of myasthenia gravis in which he found small areas of cellular infiltration between the muscle-fibres of the diaphragm and deltoid, the two muscles examined microscopically. He considered these cells resembled the small round cells present in the thymus. There was a similar collection of mononuclear cells in the heart muscle. This patient had a small tumour in the vicinity of the thymus, which was believed to be a lymphosarcoma, and the cellular infiltration in the muscles was regarded as metastases.

Mandlebaum and Celler in 1908 stated that in eleven fatal cases of myasthenia gravis about 20 per cent. showed an abnormal state of the thymus gland, while Graves's disease has occasionally occurred in combination with myasthenia gravis.

Marburg and Fujoni are reported to have found degenerative changes in the muscle fibres in the neighbourhood of the cellular deposits which they believed were a reaction to the degenerative myositis.

Buzzard, Mandlebaum and Celler regard the lymphorrhages as a true lymphocytic infiltration, and not the result of an inflammatory reaction. These lymphorrhages are derived from the blood-stream or from dilated perivascular lymph spaces. Various changes in the

muscles have been recorded, quite apart from lymphorrhages, such as a granular degeneration, swelling of the muscle-fibres and proliferation of the sarcolemma cells, and connective tissue formation. Lymphocytic infiltrations in the internal organs were described by Buzzard and these were also found in the nervous system.

Buzzard and Greenfield in discussing the morbid anatomy of myasthenia gravis state as follows: "The only characteristic feature is the presence of cellular deposits in many and various organs of the body, and particularly in the skeletal muscles." "Lymphorrhages are more commonly described in the muscles than in other organs, possibly because they have been more thoroughly investigated." They consider that the lymphorrhages are found generally in the vicinity of capillary vessels, and occur especially in the ocular muscles.

In seven out of nine cases of sudden death in young children, Rieder found an infiltration of lymphocytes and round cells in the heart muscle.

The pathological findings in the muscles in myasthenia gravis referred to in the literature are exactly similar to our findings in exophthalmic goitre, while the occasional association of the two diseases, and the fact that enlargement of the thymus gland and ocular phenomena are common to both myasthenia gravis and exophthalmic goitre, are facts which serve to link together these two diseases.

Various observers have studied control muscles, but have failed to observe similar changes to those described in myasthenia gravis, and which we have recorded in exophthalmic goitre. We ourselves have not seen such changes in the muscles in diseases other than those referred to. It is of course true that round-celled infiltration of the muscles occurs in sarcomatosis, leukæmia and lymphadenoma, but these diseases have their own distinctive feature. We hope, however, that our findings in exophthalmic goitre will result in further investigations of the muscles, especially the ocular muscles, in other diseases of the endocrine system.

Summary.

(1) Lymphorrhages have been found in the muscles in eight out of nine cases of exophthalmic goitre.

(2) They have been most marked in the ocular muscles.

(3) Lymphorrhages were both small and large. The large lymphorrhages caused wide separation of the muscle-fibres. The cell contents of the deposits were chiefly lymphocytes with some plasma and

endothelial cells. The lymphorrhages were not infrequently situated in proximity to a blood-vessel.

(4) The muscle in proximity to the lymphorrhages may show atrophic changes, while an interstitial myositis is by no means uncommon.

We are greatly indebted to our colleague, Dr. J. Bamforth, for some of the material employed for these investigations.

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SOME OBSERVATIONS ON DEEP SENSIBILITY.

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Introduction.

The researches of Head and his co-workers challenged the older views of the method of innervation of the subcutaneous tissues, since they suggested that what was termed "deep sensibility" was almost entirely dependent upon nerve-fibres which run with the motor nerves and extend to their destination along tendons. This opinion was based upon the residual sensation found after division of such a purely cutaneous nerve as the radial.

Head's work was primarily directed to a study of cutaneous (i.e., superficial) sensation, which he divided into epicritic and protopathic; but recently Stopford has investigated more fully deep sensibility. In addition to suggesting that deep sensibility ought to be subdivided, and that the subdivision of deep sensation may be dissociated, the researches of Stopford have demonstrated the difficulties of isolating the sensation of the skin or the deeper tissues. He states [3] "that no nerve to be of sufficient size to be of clinical importance, or to be selected for experimental work upon sensation, is distributed exclusively to skin. Consequently, after section of any of the nerve trunks, even in the distal part of a limb, there is bound to be some disturbance of so-called deep sensibility, and it seems impracticable to divide sensation into superficial and deep. Besides this anatomical criticism of such a division, there is to be remembered the additional confusion which may arise from the knowledge that one form of sensation at least is transmitted by both cutaneous and deep nerves."

The results, far reaching though they are, do not complete our knowledge of the subject of deep sensation, and there is still a wide field for investigation. Before discussing any further researches, it is convenient to consider in some detail our present knowledge of the different forms of sensation grouped under the term of deep sensibility.

Tactile localization.—It is probable that this form of sensation is the sum of two sets of sensation, one arising from deep and one from

cutaneous sensibility, but both Head [1] and Stopford [3] are agreed that it is possible through the deep system alone. In addition, Stopford believes that accurate localization through the deep system is disturbed by a section of the median and ulnar nerves at the wrist.

(2) *Recognition of posture and passive movement.*—Head [2] and Sherren have shown that if a part possessed deep sensibility only, the position and movements of the joints could be accurately localized. Stopford carried out a series of investigations on patients known to have a complete division of definite peripheral nerves of the upper extremity, and where there were known to be no complications, such as division of tendons, and came to the conclusion that [3] "the digital nerves are the only important source to the joints of branches which transmit impulses that enable us to recognize position and movement."

(3) *Pressure pain.*—During his investigations Stopford came to the following conclusions regarding this form of deep sensation:--

"(1) In those patients where the division of the nerve was in the distal part of the limb, and there was no absolute sensory loss, the readings on the affected side tended to be definitely lower than on the sound side.

(ii) In some patients with division of the median nerve in the middle of the forearm, that is, proximal to the supply of that portion of the flexor sublimis digitorum which flexes the index finger, and distal to the origin of the anterior interosseous nerve and branches for the supply of the main flexor mass, considerably more pressure was required on the index on the affected than on the sound side. These results pointed to a disturbance, but not complete loss, of this form of sensation in the index after division of the median, proximal to the origin of the most distal branch to the flexor sublimis digitorum, and indicated that some fibres for this form of sensation in the index were transmitted by this muscular branch and probably extended to the digit along the tendon."

It appears, therefore, that nerve fibres running with tendons may play some part in the mechanism of peripheral sensation.

(4) *Pressure (Contact).*—This is commonly the only form of sensation studied in routine clinical examinations as a means of testing for the presence or absence of deep sensibility. It is probably only affected in lesions found in the proximal parts of the limb.

Tactile discrimination has to be considered, as some observers believe it may be partly dependent upon deep innervation, although Head includes it in the epicritic subdivision of cutaneous sensation.

TABLE.

Case	Injury	Level of	Days between injury and examination	Tactile localization				Joint sense	Pressure pain				Tactile Discrimination	
				Injured		Uninjured			Injured		Uninjured		Uninjured	Injured
				Affected	Remainder	Affected	Remainder		Affected	Remainder	Affected	Remainder		
				(1)	(2)	(3)	(4)		(1)	(2)	(3)	(4)	(1)	(2)
I	Extensor longus pollicis (L)	Carpo-metacarpal joint ..	2 months	0·65	0·47	0·27	0·75	No alteration	0·20	1·80	1·20	1·60	1½ cm. 1 9 R 1 W / 2 10 R	1½ cm. 1 9 R 1 W / 2 9 R 1 W
II	Flexor carpi radialis, palmaris longus, flexor sublimis (L)	Wrist	4 days	0·36	0·50	xx 0·55	0·70	Not tested	3·98	4·50	6·36	6·50	2 cm. 1 8 R 2 W / 2 10 R	2 cm. 1 9 R 1 W / 2 10 R
III	Both flexors, index finger (L)	Metacarpo-phalangeal joint	6 weeks	0·33	0·23	x 0·26	0·33	No alteration	2·66	8·62	7·25	8·31	3 cm. 1 7 R 3 W / 2 9 R 1 W	3 cm. 1 7 R 3 W / 2 9 R 1 W
IV	Extensor communis, middle finger (L)	Metacarpo-phalangeal joint	3 weeks	0·88	0 73	0 60	0·76	Not tested	0·25	3·25	3·30	3·75	2 cm. 1 10 R / 2 6 R 4 W	2 cm. 1 10 R / 2 2 R 8 W
V	Extensor communis, little, ring and middle fingers (L)	1 in. proximal metacarpo-phalangeal joint	8 days	0·44	0·32	x 0·35	0·30	Not tested	2·75	4·20	4·10	4·60	2½ cm. 1 7 R 3 W / 2 5 R 5 W	2½ cm. 1 7 R 3 W / 2 10 R
VI	Extensor communis, ring and middle fingers (L)	Carpo-metacarpal joint ..	4 days	x 0·55	0·73	0·46	0·45	Not tested	0·00	4·25	5·46	6·50	2 cm. 1 9 R 1 W / 2 10 R	2 cm. 1 7 R 3 W / 2 10 R
VII	Extensor communis digitorum, all (L)	Carpo-metacarpal joint ..	2 days	x 0·38	0·57	x 0·43	0·50	Not tested	1·75	3·50	2 87	3·25	2 cm. 1 9 R 1 W / 2 10 R	2 cm. 1 8 R 2 W / 2 10 R
VIII	Extensor communis digitorum, index fingers (L)	Carpo-metacarpal joint ..	4 days	0·22	0·28	0·38	0·18	Not tested	1·16	4·25	3·80	4·33	2 cm. 1 10 R / 2 7 R 3 W	2 cm. 1 10 R / 2 8 R 2 W
IX	Flexor sublimis (all), palmaris longus (L)	Wrist	2 days	xxx 0·38	0·20	xx 0·33	0·30	No alteration	1·50	4·75	4·52	4·33	1 cm. 1 10 R / 2 10 R	1 cm. 1 9 R 1 W / 2 10 R
X	Extensor ossis, metacarpi pollicis (L)	1 in. distal to wrist ..	7 days	0·50	xx 0·40	0·30	xxx 0 27	Not tested	1·18	4·50	4·38	4·75	2 cm. 1 8 R 2 W / 2 10 R	2 cm. 1 3 R 7 W / 2 9 R 1 W
XI	Flexor longus pollicis (L) ..	Carpo-metacarpal joint ..	2 days	0·36	0·60	0·44	0·65	Not tested	0·75	5·00	4·66	5·00	1½ cm. 1 9 R 1 W / 2 9 R 1 W	1½ cm. 1 8 R 2 W / 2 10 R
XII	Extensor longus pollicis (L)	Wrist	3 days	0·38	0·38	0·45	0·52	Not tested	2·38	5·13	5·75	4·75	2½ cm. 1 10 R / 2 10 R	2½ cm. 1 4 R 6 W / 2 7 R 3 W
XIII	Flexor sublimis digitorum, little finger (R)	Wrist	2 days	0·37	0·96	0·48	0·96	Not tested	0·67	2·65	1·67	2·75	1½ cm. 1 10 R / 2 10 R	1½ cm. 1 10 R / 2 10 R
XIV	Flexor longus pollicis (R) ..	Metacarpo-phalangeal joint	10 weeks	0·42	0·20	0·58	0·30	No alteration	2·13	4·63	3·88	5·25	1½ cm. 1 10 R / 2 10 R 1 W	1½ cm. 1 10 R / 2 10 R
XV	Extensor communis digitorum, middle finger (R)	½ in. proximal metacarpo-phalangeal joint	5 weeks	0·40	x 0·35	1·00	0·52	(?) Slight alteration	2·80	3·30	3·91	3·41	2½ cm. 1 9 R 1 W / 2 9 R 1 W	2½ cm. 1 7 R 3 W / 2 7 R 3 W
XVI	Flexor carpi radialis, flexor longus pollicis (R)	Wrist	5 days	0·44	0·25	0·77	0·20	Not tested	3·00	5·13	5·15	5·38	2½ cm. 1 10 R / 2 9 R 1 W	2½ cm. 1 10 R / 2 10 R
XVII	Flexor carpi ulnaris, flexor carpi radialis, flexor longus pollicis (L)	Wrist	5 days	0·14	0·15	0·30	0·05	Not tested	4·60	4·83	6·55	5·33	1½ cm. 1 10 R / 2 10 R	1½ cm. 1 10 R / 2 10 R
XVIII	Flexor longus pollicis (R) ..	Metacarpo-phalangeal joint	18 days	0·28	x 0·21	xxxx 0·47	0·18	No alteration	2·75	5 91	5·16	5·66	2 cm. 1 10 R / 2 10 R	2 cm. 1 9 R 1 W / 2 5 R 5 W
XIX	Extensor communis digitorum, middle finger (L)	Carpo-metacarpal joint ..	7 days	0·30	0·36	0·20	0·33	Not tested	0·58	5·91	5 91	5·81	1½ cm. 1 10 R / 2 10 R	1½ cm. 1 5 R 5 W / 2 10 R
XX	Flexor profundus digitorum, flexor sublimis digitorum (middle finger, R)	Metacarpo-phalangeal joint	4 days	0·30	0·26	0 40	0·23	Not tested	0·92	5·33	5 00	5·75	1½ cm. 1 10 R / 2 10 R	1½ cm. 1 9 R 1 W / 2 9 R 1 W
	Averages	.. .. ..	.. ..	0·40	0·41	0·45	0·42	..	1·81	4 57	4·54	4·85	Totals	 16

Tactile localization expressed in centimetres.
x = Wrong localization.

Pressure pain expressed in kilogrammes.

TABLE.

Tactile localization			Joint sense	Pressure pain				Tactile Discrimination						Remarks
ured	Uninjured			Injured		Uninjured		Uninjured	Injured	Uninjured		Injured		
Remainder	Affected	Remainder		Affected	Remainder	Affected	Remainder			1 point	2 points	1 point	2 points	
(2)	(3)	(4)		(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)	(5)	(6)	
0·47	0·27	0·75	No alteration	0·20	1·80	1·20	1·60	1½ cm. 1 9 R 1 W / 2 10 R	1½ cm. 1 9 R 1 W / 2 9 R 1 W	1	0	1	1	..
0·50	xx 0·55	0·70	Not tested	3·98	4·50	6·36	6·50	2 cm. 1 8 R 2 W / 2 10 R	2 cm. 1 9 R 1 W / 2 10 R	2	0	1	0	..
0·23	x 0·26	0·33	No alteration	2·66	8·62	7·25	8·31	3 cm. 1 7 R 3 W / 2 9 R 1 W	3 cm. 1 7 R 3 W / 2 9 R 1 W	3	1	3	1	Unsuccessful suture
0 73	0 60	0·76	Not tested	0·25	3·25	3·30	3·75	2 cm. 1 10 R / 2 6 R 4 W	2 cm. 1 10 R / 2 2 R 8 W	0	4	0	8	A bad subject for testing
0·32	x 0 35	0·30	Not tested	2·75	4·20	4·10	4·60	2½ cm. 1 7 R 3 W / 2 5 R 5 W	2½ cm. 1 7 R 3 W / 2 10 R	3	5	3	0	..
0·73	0·46	0·45	Not tested	0·00	4·25	5·46	6·50	2 cm. 1 9 R 1 W / 2 10 R	2 cm. 1 7 R 3 W / 2 10 R	1	0	3	0	..
0·57	x 0·43	0·50	Not tested	1·75	3·50	2 87	3·25	2 cm. 1 9 R 1 W / 2 10 R	2 cm. 1 8 R 2 W / 2 10 R	1	0	2	0	..
0·28	0·38	0·18	Not tested	1·16	4·25	3·80	4·33	2 cm. 1 10 R / 2 7 R 3 W	2 cm. 1 10 R / 2 8 R 2 W	0	3	0	2	..
0·20	xx 0·33	0·30	No alteration	1·50	4·75	4·52	4·33	1 cm. 1 10 R / 2 10 R	1 cm. 1 9 R 1 W / 2 10 R	0	0	1	0	Interruptions during examination
xx 0·40	0·30	xxx 0 27	Not tested	1·38	4·50	4·38	4·75	2 cm. 1 8 R 2 W / 2 10 R	2 cm. 1 3 R 7 W / 2 9 R 1 W	2	0	7	1	Interruptions during examination
0·60	0·44	0·65	Not tested	0·75	5·00	4·66	5·00	1½ cm. 1 9 R 1 W / 2 9 R 1 W	1½ cm. 1 8 R 2 W / 2 10 R	1	1	2	0	..
0·38	0·45	0·52	Not tested	2·38	5·13	5·75	4·75	2½ cm. 1 10 R / 2 10 R	2½ cm. 1 4 R 6 W / 2 7 R 3 W	0	0	6	3	..
0·96	0·48	0·96	Not tested	0·67	2·65	1·67	2·75	1½ cm. 1 10 R / 2 10 R	1½ cm. 1 10 R / 2 10 R	0	0	0	0	..
0·20	0·58	0·30	No alteration	2·13	4·63	3·88	5·25	1½ cm. 1 10 R / 2 9 R 1 W	1½ cm. 1 10 R / 2 10 R	0	1	0	0	Tendon not sutured
x 0·35	1·00	0·52	(?) Slight alteration	2·80	3·30	3·91	3·41	2½ cm. 1 9 R 1 W / 2 9 R 1 W	2½ cm. 1 7 R 3 W / 2 7 R 3 W	1	1	3	3	Tendon not sutured
0·25	0·77	0·20	Not tested	3·00	5·13	5·15	5·38	2½ cm. 1 10 R / 2 9 R 1 W	2½ cm. 1 10 R / 2 10 R	0	1	0	0	Tendon not sutured
0·15	0·30	0·05	Not tested	4·60	4·83	6·55	5·33	1½ cm. 1 10 R / 2 10 R	1½ cm. 1 10 R / 2 10 R	0	0	0	0	Tendons cut doubtful. Median and ulnar not involved
x 0·21	xxxx 0·47	0·18	No alteration	2·75	5 91	5·16	5·66	2 cm. 1 10 R / 2 10 R	2 cm. 1 9 R 1 W / 2 5 R 5 W	0	0	1	5	No history of injury to other thumb; not sutured
0·36	0·20	0·33	Not tested	0·58	5·91	5 91	5·81	1½ cm. 1 10 R / 2 10 R	1½ cm. 1 5 R 5 W / 2 10 R	0	0	5	0	..
0·36	0 40	0·23	Not tested	0·92	5·33	5 00	5·75	1½ cm. 1 10 R / 2 10 R	1½ cm. 1 9 R 1 W / 2 9 R 1 W	0	0	1	1	..
0·41	0·45	0·42	..	1·81	4·57	4·54	4·85	Totals ..	.. ..	16	17	38	25	..

↑ ation expressed in centimetres. Wrong localization.

↑ Pressure pain expressed in kilogrammes.

The investigations described in this paper were undertaken on the suggestion of Professor Stopford in an endeavour to throw further light on some points in connection with sensation which still remain obscure. So far all the researches on peripheral sensation have been performed on patients known to have a lesion of some peripheral nerve, and consequently it seemed that information might be obtained from the systematic examination of patients suffering from division of tendons in the forearm. From a few preliminary experiments it became apparent that patients exhibiting an uncomplicated tendon division only could be of service, and all others were discarded. By this means it should be possible to define the extent of the innervation of deep tissues by nerve fibres transmitted by the tendon, since these would be almost inevitably divided, and there was no involvement of any other nerve path. Careful testing of cutaneous sensation was sufficient to exclude damage to nerve fibres other than those accompanying the tendons. The tests used to examine the various forms of sensation were those described in papers by Head, with the minor alterations suggested by Stopford.

In all twenty patients were examined: the comparatively small number is accounted for by the fact that even in large centres uncomplicated tendon lesions are by no means common, and it has taken very considerable time to collect even this number.

A brief survey of the results reveals two facts:—

(1) Division of a tendon at any point between the wrist and the metacarpo-phalangeal joints produces a similar result as far as sensation is concerned, and therefore no significance can be attached to the level of the injury to the tendon between these points.

(2) Regeneration of nerve fibres associated with tendons is, in common with regeneration of nerve fibres elsewhere in the body, a slow process; there is no evidence of any modification in the findings dependent on this factor, though the time between the date of injury and date of examination has varied from two days to ten weeks.

Summary of Results.

It is striking that in at least seventeen of the twenty patients examined the findings were almost identical. For convenience the results of the tests of the various forms of sensation will be considered separately.

Tactile localization.—The average error in localization for the series

in the "affected" part (i.e., the part of the hand towards which the affected tendon is running) was 0·40 cm. and in unaffected parts of the same hand 0·41 cm. These two figures are for practical purposes identical. The criticism that it is not possible to define the affected region after tendon division, and that these figures lose their force as nerve fibres may be distributed to all parts of the hand, may be met by reference to the examination of the other hand. The average error on the sound side was 0·43 cm., the slightly higher reading being possibly due to the fact that the sound side was invariably tested first. These figures at first sight seem to suggest that tactile localization is not dependent on nerve fibres transmitted by tendons, but it must be remembered, as Head has pointed out, "that this form of sensation is the sum of two sets of sensations, one arising from deep, and one from cutaneous sensibility." Therefore it can only be said that loss of these fibres does not affect tactile localization, and that this form of sensation cannot be to any great extent dependent upon them.

Recognition of passive movement and posture.—In fourteen patients it was unfortunately impossible to test this form of sensation, but in five of the six cases in which it was done no variation was noted. In the remaining patient a slight subjective alteration was noted on comparing the two sides. This suggests that nerve fibres associated with tendons are at the most only very slightly concerned with this form of sensation, and helps to confirm Stopford's observations [3] that "the digital nerves are the only important source to the joints of branches which transmit impulses that enable us to recognize position and movement."

Pressure pain.—Taking all the cases together, the average amount of pressure required to produce pain over the affected part (i.e., that part towards which the affected tendon is running) of the injured side is found to be 1·81 kgm. The average amount of pressure required to produce pain over the remainder of the injured side is 4·57 kgm. There is a marked difference between the two sets of figures, and this is as noticeable when the figures are compared with those of the uninjured side: here the corresponding readings are 4·54 and 4·85 kgm. respectively. In the normal individual (i.e., where no traumatic or pathological lesion is present) the four sets of figures would approximately correspond.

Reference to the examination of pressure pain in the accompanying table shows that consistently lower readings were obtained in the region of the insertion of the divided tendon or tendons when compared with

those obtained on testing other regions on the same hand, or identical points on the unaffected side. The explanation of these observations is not easy and must not be considered to denote that division of tendons has interfered directly with pressure pain, because if some of the fibres concerned with this element of sensation had been divided, the readings found in the affected region would have been higher and not lower than those in normal parts. In the light of modern work upon sensation it would appear that the explanation is to be found in a defect in some form of sensation other than pressure pain. The response to less pressure is most probably due to a loss of a number of tactile pressure fibres, and would constitute a "release" phenomenon. Under normal circumstances pressure from the algometer will stimulate the tactile pressure end-organs first, and pressure pain end-organs only when the pressure is adequate for this purpose, and, as Head has stated, the development of the painful sensation is gradual and emerges out of a mixed general sensibility. After loss of an appreciable number of tactile pressure fibres, the appreciation of pain appears quickly and seems to account for lower readings. Anyone familiar with the use of the algometer in peripheral nerve lesions will, from the behaviour of the patient, recognize the two different types of appearance of the painful element in the sensation, and the descriptions elicited from the more intelligent patients are most convincing and support in every way Head's own experiences. Therefore, it seems highly probable that the findings when using the algometer show a disturbance of tactile pressure and not of pressure pain. It was not possible to confirm this by testing with graduated hairs, since in the patients under review in this paper cutaneous sensation was unaffected, and the lightest possible stimulus was in consequence appreciated at once. The observations made upon pressure pain also presented some evidence that nerve fibres associated with tendons innervate chiefly those deep structures which are in the immediate neighbourhood of the insertion of the tendon.

Tactile discrimination.—Here the findings are somewhat confusing. The total mistakes made in the series yield the following results:—

On the uninjured side, one stimulation was wrongly noted sixteen times, and two simultaneous stimulations were wrongly noted twenty-five times.

On the injured side, one stimulation was wrongly noted thirty-eight times and two simultaneous stimulations were wrongly noted twenty-five times.

Turning to individual cases, confusion becomes worse: some cases

show no error on either side (Cases XIII and XVII) : some show more error on the uninjured than on the injured side (Cases II, V, VIII, XIV, and XVI) : while others show more errors on the injured than on the uninjured side (notably Cases IV, X, XII, XV, XVIII and XIX). From these results it can only be said that there is no definite evidence that the fibres accompanying the long tendons are concerned with tactile discrimination.

Conclusions.

(1) It has been shown that nerve fibres which run to their distribution in the subcutaneous tissues along tendons are concerned with deep sensibility, but not so profoundly as believed by Head.

(2) Division of these fibres does not result in any serious loss of sensation, tactile pressure alone being affected to some extent in most cases. A few of these fibres may rarely be concerned with the conduction of sensory impulses from joints, but there is no evidence to show that the power of localization or of discrimination is dependent upon them.

(3) The fibres appear to follow the tendons to their insertion where they are chiefly distributed to the neighbouring deep tissues.

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ABSCESS OF THE SPINAL CORD: REPORT OF A CASE WITH FUNCTIONAL RECOVERY AFTER OPERATION.[1]

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ABSCESS of the spinal cord is not common. A searching review of the literature disclosed reports of but twenty-nine cases; in all except one of these the disease terminated fatally (tabulation). In 1899, Cavazzani reported that a woman had been cured by operation; she, however, remained totally paralysed below the seat of the lesion.

REPORT OF A CASE.

November 30, 1923, a Finnish girl, aged 11, was brought to the Mayo Clinic because of progressive weakness of the lower extremities. In June, 1921, she had been chilled while picking berries, and the same evening she had a little fever, but slept well during the night. In the morning she discovered that she could not get out of bed because both legs were almost completely paralysed. For the next two weeks her temperature remained between 100° and 101° F. It was necessary to empty the bladder by catheter, and control of urination was greatly impaired. During the third month she observed that her legs were growing stronger and she was able to leave her bed. Improvement continued and she returned to school in October. She thought that she could walk as well as the other children, but could run only half as well. She did not recall that sensation had suffered any impairment during her illness. No further change took place until May, 1923, when the legs became weaker; the weakness continued steadily, so that by August she found it necessary to assist herself with her hands whenever she wished to arise from a chair; by September she was quite unable to walk. In October sensation also became impaired. At no time had there been pain.

Examination disclosed slight redness of the optic discs; the retinal veins were full and tortuous. The tonsils were large and infected. The temperature was 99·4° F., and the leucocyte count 12,500. A decubital scar, which had appeared in the course of the patient's illness, in 1921, was present over the sacral area. Both legs were spastic and almost completely paralysed. If the patient raised her head while recumbent the umbilicus moved sharply upward. The abdominal reflexes were absent, the patellar and

[1] Read before the Minnesota Neurological Society and before the Society of Neurological Surgeons, September 29, 1925, St. Paul, Minnesota, and November 6 and 7, 1925, Rochester, Minnesota, respectively.

tendo Achillis reflexes were markedly increased, and the plantar responses strongly extensor by Babinski's and Chaddock's methods. Touch, pain and temperature sensibilities were greatly impaired over the lower limbs and peri-anal area; the sensory disturbance faded rapidly over the abdomen, but a level, which corresponded to about the eighth dorsal segment, could be established. Vibratory and postural sensibilities were absent in the legs. The left

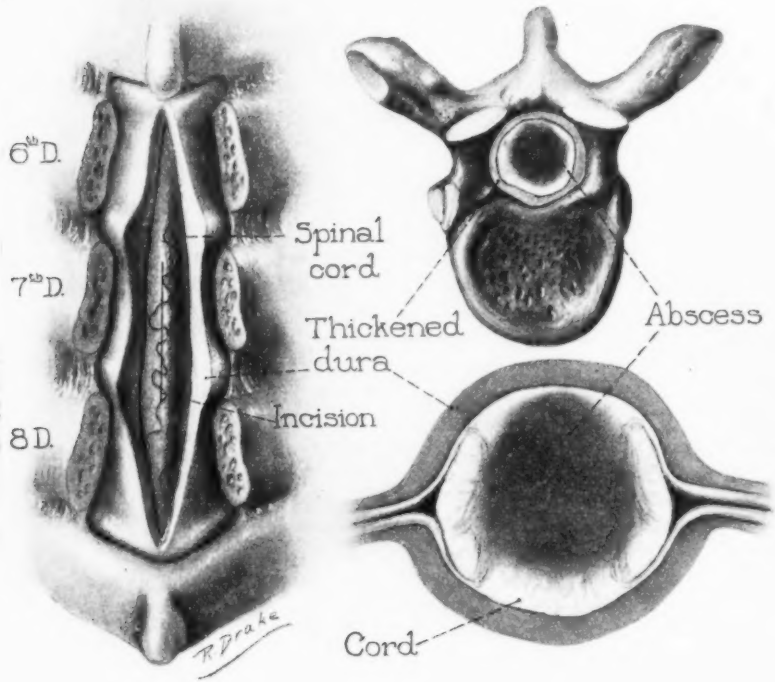

FIG. 1.—The thickness of the dura, the bulging of the dura between the laminæ, and the involvement of the cord, which fills the entire spinal canal, are shown.

leg felt warm to the touch, the right leg was pale and cold. Control of the sphincters was considerably impaired. Only 4 c.c. of spinal fluid could be obtained; it was yellow and under very low pressure, which was not altered by compression of the jugular veins. Nonne's test was positive, and there were two small lymphocytes to the cubic millimetre.

A diagnosis of meningomyelitis with spinal block was made. While the attack of paralysis in 1921 suggested poliomyelitis, because of the incon-

tinence, the decubitus, and the residual spasticity, we believed that it was due to an acute myelitis. The return of symptoms was interpreted as a result of a lighting up of the old process, or a reinfection at the seat of the former lesion. A similar course is not infrequently noted in cases of brain abscess.

Owing to the progression, the establishment of a level and the evidence of block, it was thought advisable to explore, although the uniform involvement of all qualities of sensation, the profound peri-anal sensory disturbance, to which Kerppola called attention as indicating an intramedullary lesion, the absence of pain, and the belief that we were dealing with an infectious process, did not promise a very satisfactory result.

December 31, 1923, laminectomy of the sixth, seventh and eighth dorsal vertebræ was performed. The dura bulged between the laminæ, was found to be about 4 mm. in thickness throughout its entire circumference and length of exposure, and presented a beaded appearance (fig. 1). This condition extended upward beyond the visible field, and caused the cord to be compressed. On incising the cord along the posterior median septum, the fluid contents of an encapsulated abscess, about 1·5 cm. in length, escaped. Because of the grave involvement of the cord the prognosis at this time was: "The patient will never walk." On microscopic examination the fluid was revealed to be pus; no organism could be found on staining it by Gram's and Ziehl-Neelson's methods; there was no growth on blood agar, but a slight growth of staphylococci appeared in dextrose broth; inoculation of two guinea-pigs with the pus did not cause infection.

A return of sensation was noted and movement of the limbs was possible within two weeks after operation. The patient was dismissed from the hospital on January 30, 1924. By March she was able to walk about the house without crutches. Improvement continued until June 1, when a relapse occurred. Examination July 8 revealed pronounced weakness with spasticity, increased tendon reflexes below, and abnormal reflexes of the toes, particularly on the left. The girl could walk a little with assistance. Sensation, however, showed marked improvement for all qualities, there being only slight impairment below the knees. Control of the sphincters was normal. No recommendations were offered except massage. In view of the subsequent improvement this relapse could not be explained.

The patient later went under the care of Dr. Ernest Hammes, of St. Paul, and made a remarkable recovery. Through his courtesy we were permitted to present her at the meeting of the Minnesota Neurological Society, September 29, 1925. She could arise from her chair without apparent effort or hesitation; she could walk with ease and alacrity, not only in the usual manner, but also on her toes and on her heels; she could stand on either foot alone. It was only when she tripped over a rug and was recovering her equilibrium that a slight ataxia was betrayed. Sensation was practically intact. The patellar and tendo Achillis reflexes were normal; the plantar responses were uncertain.

Discussion.

Although much discussion might be indulged in, this pleasure is left to the reader. The first case of cord abscess was probably that reported by Hart in 1830. Brera, in 1810, reported three cases, thought by him to have been cord abscesses; however, Ullmann cites Abercrombie as having questioned the diagnoses in these cases as early as 1835, stating that they were probably cases of softening. We

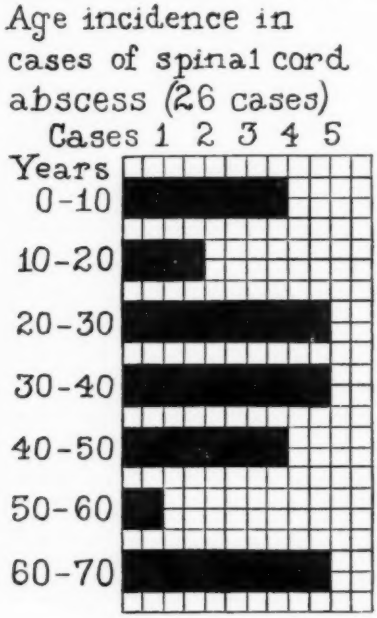

Fig. 2.

were unable to obtain Brera's original article or abstracts of his case reports.

That persons of any age may be affected by cord abscess is shown by fig. 2. The youngest patient reported was 8 days old, and the eldest 61 years. Twenty were males, and eight were females; the sex of one patient was not given. These figures suggest that the male is more susceptible to cord abscess than the female, but the cases are too few to draw this conclusion unreservedly.

Causes to which the abscess formation has been ascribed are presented in fig. 3. It is seen that trauma plays a prominent part. Some interesting experiments in this regard were carried out by Hoche. He

introduced aseptic emboli, of pollen, starch, clay, air, and croton and castor oil, into the aortas of rabbits and dogs by means of a long

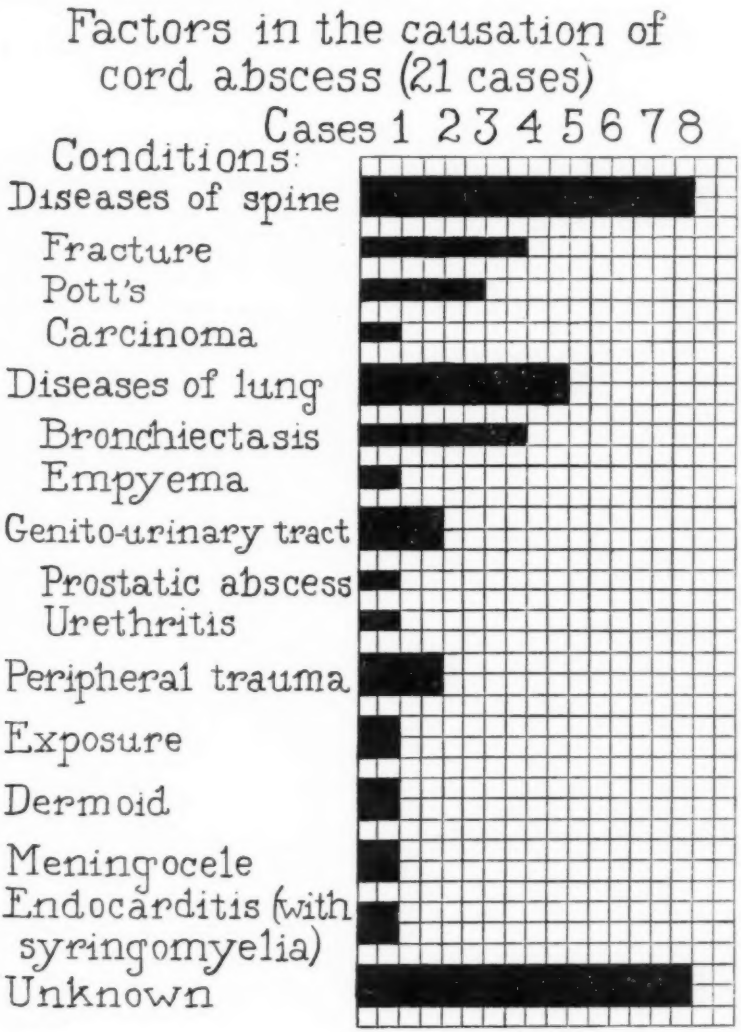

FIG. 3.

cannula. He found that infarction of the cord could be produced only when the emboli were sufficiently small to enter the intramedullary

vessels. The grey matter was involved more extensively than the white. When staphylococci, pneumococci, or *Bacillus coli* only were introduced, infarction and abscess formation rarely took place, but when these organisms were introduced along with aseptic emboli, which injured the vessels of the cord, abscess formation was common. Hoche concluded that trauma and chilling were important agents in predisposing to infections of the spinal cord.

The low incidence of abscess of the cord is attributed by various writers to: (1) the relatively small mass of the cord (the ratio of its volume to that of the brain is given as 2 : 100) [29]; (2) the small lumen and indirect course of the blood-vessels entering the spinal cord; (3) the protected location of the cord as compared to the brain; and (4) a protective agent, probably chemical. On what facts Schlesinger bases this hypothesis he does not state. Nothnagel believes that some agent associated with infections of the lung renders the central nervous system less resistant to infection.

The disease is serious. Its course is as a rule foudroyant; the shortest was three days, and half of the patients died within two weeks. The longest duration was seven months (fig. 4).

The involvement of the cord was diffuse in twenty-two cases; it was focal (three segments or less) in six; there were multiple cord abscesses in three; brain abscess was associated with the cord lesion in four. No level of the cord was exempt, none was predisposed. Meningitis was associated in at least fifteen cases; it was definitely reported as absent in two. The diffuse involvement and fulminant course probably explain the flaccid paralysis and absence of the tendon reflexes observed in at least half the cases.

The invading organism was demonstrated in thirteen cases. Streptococci were found in five cases, with Gram-negative bacilli and staphylococci associated in one each; staphylococci were found in four cases, with streptococci and another bacillus associated; actinomycosis was demonstrated in four, with pneumococci associated in one, and short bacilli were found in one.

A diagnosis of cord abscess can rarely be made. This honour belongs to Nothnagel, who made the diagnosis in a patient who also had bronchiectasis, basing his opinion on experience gained from cases of brain abscess complicating bronchiectasis; and to Homén, who had himself observed a patient with cord abscess previously. Abscess of the cord is rarely considered in making a diagnosis of a condition that resembles localized meningomyelitis. The presence of spinal block and

a yellow fluid may aid in deciding on the course to be taken. Should the diagnosis be correct few patients would be saved by exploration, since the lesion is usually diffuse. Still, it is certain that patients

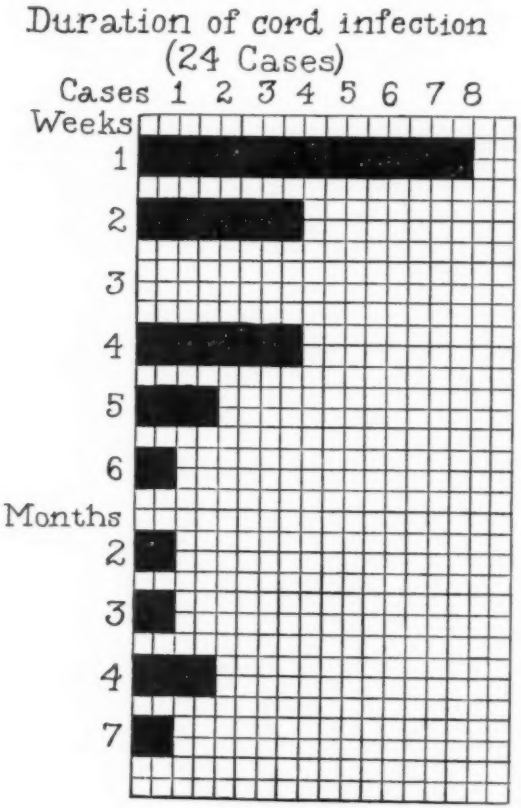

FIG. 4.

having cord abscesses will be saved from time to time, but probably only through venturing on exploratory laminectomy and incision of the cord.

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(*Continued on page* 206.)

TABULATION.

ABSTRACTS OF TWENTY-NINE CASES OF SPINAL CORD ABSCESS REPORTED IN THE LITERATURE

Author, year	Sex, age years	Primary cause	History	Duration	Outcome	Necropsy	Location of abscess	Organism	Remarks
Hart, 1830	? 0·75	..	Convulsions. Symptoms of hydrocephalus	..	Died	Internal hydrocephalus; pus in hydromyelic canal. Cyst to right of cauda	First to twelfth dorsal	..	..
Ollivier, 1837	M. 23	Fracture, spine	Fell; legs paralysed, arms weak. Return of power and sensation above pubis and in right leg. Decline	15 days	Died	Fracture, seventh cervical vertebra. Cord abscess	Seventh to eighth cervical	..	..
Ollivier, 1837	M. 60	Fracture, spine	Fell from tree; legs paralysed; incontinence	1 month	Died	Fracture, tenth dorsal. Cord abscess "size of hazel-nut"	Tenth dorsal	..	..
Fairbrother, 1852	M. 60	Exposure	Worked in water; same night, retention of urine, loss of power and sensation in legs	..	Died	Cord abscess "size of bean." Meninges congested	Eighth dorsal	..	..
Jaccoud, 1864	M. 32	..	Backache, fever; same evening complete paraplegia	10 days	Died	Suppurative myelitis ..	Eighth dorsal down	..	..
Demme, 1875	F. 3·5	Poliomyelitis?	Fever; paralysis same night; bronchopneumonia	4·5 months	Died	Miliary abscesses in anterior horns and column, lumbar enlargement. Atrophy anterior horn cells, cervical enlargement	Lumbar enlargement	..	Diagnosis; paralysis essentialis infantilis
Feinberg, 1876	M. 60	Fracture, spine	While carrying weight fell on buttocks; immediate loss of power and sensation below navel; incontinence; tendon reflexes absent in legs; deformity first lumbar. Pneumonia	27 days	Died	Fracture, first lumbar, decubitus. Cord abscess	Below first lumbar vertebra	..	..
Nothnagel, 1884	M. Adult	Bronchiectasis	Girdle pain in abdomen; dysuria; progressive loss of power; sensation and reflexes below the epigastrium	5 days	Died	Meningitis of brain and cord. Multiple abscesses in centrum semi ovale. Malodorous cord abscess	From fifth dorsal down	..	Cord abscess diagnosed because of associated bronchiectasis

Dubreuilh, 1886	F. 2·5	Dermoid	Eight months before nævus-like swelling from sixth to seventh cervical spine, which discharged pus for five months; torticollis for six weeks; progressive tetraplegia for four months with epileptoid tremors and increased tendon reflexes; bronchopneumonia	4 months	Died	Dermoid containing fat, smooth muscle, sweat glands, hair; tract through dura at third cervical; pachymeningitis; abscess mostly in central grey and right posterior horn. Bronchopneumonia	Medulla to left lumbar cord	..	..
Ullmann, 1889	M. 27	Gonorrhœal urethritis	Dysuria, backache. Loss of motion and sensation in legs in four days. Ascending paralysis from seventh to fourth dorsal. Stiff neck	27 days	Died	Meningitis; two cord abscesses	Cervical and lumbar cords	..	Thought to be three metastatic lesions from urethra; one to pia and two to cord. Not gonococcus but secondary invader
Eisenlohr, 1892 (Case 1)	M. 43	Empyema	Seven weeks before. resection empyema. Three days before, while washing cavity, sudden weakness in left arm; progressive loss of power and sensation in left leg and arm, then right leg and arm	3 days	Died	Abscess "size of pea," left ala cinerea and cord partly encapsulated	Medulla and pons to second cervical	Short bacillus	..
Schlesinger, 1894	M. 31	Injury, perineum; prostatic abscess	Fell two and a half months before, injured perineum; chill three hours later; dysuria. Ten days later prostatic abscess drained. Six weeks before death—headache, stiff neck, vomiting; improved. Twenty-two days before—right hand weak, paroxysmal tremor. Eighteen days before — right leg paralysed, left weak; incontinence. Sensation normal; knee jerks absent	6 weeks	Died	Abscess "size of half a pea" at decussation pyramidum. Medulla soft; abscess "size of bean"; left posterior horn and column, cervical enlargement; meningitis	Pyramidal decussation, middle cervical to fourth dorsal	Staphylococcus	Meningitis with abscess spreading into cord through posterior horn
Homén, 1895	M. 56	Bronchiectasis	Eight days before—pain in chest, shoulders and legs. Six days before—sudden paralysis of left leg. Progressive loss of power and sensation, both legs	6 days	Died	Bronchopneumonia. Multiple cerebral abscesses. No meningitis. Cord abscess, chiefly posterior and lateral columns	Fourth cervical to eighth dorsal	Streptococcus	..

ABSTRACTS OF TWENTY-NINE CASES OF SPINAL CORD ABSCESS—*Continued.*

Author, year	Sex, age, years	Primary cause	History	Duration	Outcome	Necropsy	Location of abscess	Organism	Remarks
Skála, 1896	M. 22	Gangrenous lung	Chronic productive cough. Four days before—severe cough, foul sputum containing lung tissue. Sleepless; pain in head and spine. Temperature, 103·1° to 107·8° F. Coma	6 days	Died	Gangrene of left lung. Meningitis. Pus and hæmorrhages over 3 cm. of cord	Upper dorsal	..	Diagnosis: gangrenous lung, meningitis
Schlesinger, 1897	F. 28	Caries	Sudden loss of power and sensation in legs	9 weeks	Died	Pneumonia, meningitis of brain and cord; partly encapsulated abscess, grey and ventral column	Lumbar and sacral cord	Staphylococcus	..
Cavazzani, 1899	F. 18	Fracture, spine	Fell from window thirty-nine days before. Kyphos twelfth dorsal spine; loss of power and sensation below groin; retention. Decubitus, progressive failure. Septic temperature appeared eleven days before operation. Laminectomy; thickened membranes, intramedullary abscess opposite twelfth dorsal spine. Inspissated pus, difficult to remove. Recovered from fever and decubitus. Residual loss of sensation, flaccid paralysis with atrophy of legs; involuntary; angioparalytic œdema	11 days (?)	Alive, paraplegic	.. ..	Twelfth dorsal	..	Trauma of cord; secondary hæmatogenous infection of clot
Chiari, 1900	M. 43	Bronchiectasis	Four months before, peritonitis. Three days before, pain in neck and right half of body; progressive paralysis, right side. Diagnosis: probable cerebral hæmorrhage	? 3 days	Died	Bronchiectasis; cerebrospinal meningitis; ventricular fluid cloudy; left cerebellar abscess; cord abscesses, especially in ventral part of posterior and lateral columns	Lower end of medulla, and entire cervical cord, third and fourth dorsals, and lower half of dorsal cord	Diplococcus pneumoniæ. Actinomyces	Diagnosis: probable cerebral hæmorrhage

Silfvast, 1901	M. 30	..	Eighteen days before, headache; completely blind in three days. Fundus negative; four days before, paralysis of left leg; developed Brown-Séquard syndrome; progressive paralysis of motion and sensation in both legs; lost tendon reflexes	? 4 days	Died	Apical infection; tuberculosis of bowel; cord abscess	Fourth cervical to upper dorsal	Streptococci	Cord abscess correctly diagnosed by Homén because of associated retrobulbar neuritis
Pribitkoff and Malolietkoff, 1901	F. 30	..	Twenty-three days before, fever and headache for one week. Eleven days before, girdle pain about waist, same night paralysis of right foot, and next day of left foot. Retention. Absent patellar reflexes. Anæsthesia ascended from navel to armpits. Nuchal and occipital pain	11 days	Died	Spinal leptomeningitis. Extensive cord abscess of grey and white matter	Second dorsal to conus	Actinomycosis	Believed meningitis and cord abscess started independently. Diagnosis: acute ascending myelitis
Cassirer, 1902	F. ?	Endocarditis	Six years painless infection and loss of fingers of left hand. Eight days of fever and rapidly became very ill. Loss of pain and temperature sensations over arms and trunk	8 days	Died	Recent endocarditis; pyelonephritis; multiple infarcts in lungs, spleen, kidneys, and cord; cervical and dorsal syringomyelia. Multiple abscesses not over 1 mm. in size, containing blood, small round cells and pus cells, mostly in grey, also in white matter; none noted in syringomyelic portion of cord	Multiple upper cervical and lower dorsal and lumbar	..	Considers emboli from endocarditis as cause of cord abscess
Turner and Collier, 1904	F. 61	Carcinoma of spine	Pain in shoulders for one year; ten months' onset, progressive spastic paralysis in legs and weakness and atrophy of hands; loss of sensation below seventh cervical. Tendon reflexes increased. Highest temperature 100°	?	Died	Carcinoma from third to seventh cervical vertebræ, with compression of eighth cervical and first dorsal segments. Walled-off abscess	Fourth to seventh cervical	Staphylococci and streptococci	..

ABSTRACTS OF TWENTY-NINE CASES OF SPINAL CORD ABSCESS—*Continued.*

Author, year	Sex, age, years	Primary cause	History	Duration	Outcome	Necropsy	Location of abscess	Organism	Remarks
Turner and Collier, 1904	F. 37	Pott's disease	Pain in lower spine for eleven months. One month, sudden paralysis below third dorsal. Lost tendon reflexes. Temperature 101° to 105° F. Deformity at ninth dorsal	1 month	Died	Tuberculous disc between ninth and tenth dorsals. Cord abscess and suppurative myelitis	First dorsal with scattered involvement from third cervical to conus	..	..
Turner and Collier, 1904	M. 17	Pott's disease	Kyphosis for nine years. Three months before, sudden flaccid paralysis below eighth dorsal; tendon reflexes lost. Few pus cells and 1 per cent. albumin in spinal fluid. Temperature 102° to 103° F. Ascent of level to second dorsal. Temporary improvement. Pneumonia	3 months	Died	Abscess, posterior column cord	Sixth cervical to seventh dorsal	..	..
Wolff, 1909	M. 8 days	Meningitis	Lumbar meningocele ruptured at birth. Repair	8 days	Died	Pemphigus, meningitis, unencapsulated cord abscess, mostly in posterior column	Seventh dorsal to fourth lumbar	..	..
Kawashima, 1910	M. 35	..	One month before, loss of vision of right eye and then left; numbness of right leg and sphincter disturbance. Progressive course: seven days complete paralysis below nipples, four days left arm numb; bloody spinal fluid	1 month	Died	Meningitis; cord abscess chiefly posterior horns and columns	Upper dorsal to lumbar	..	Onset with retrobulbar neuritis. Diagnosis: lead palsy

Kawashima, 1910	M. 42	..	Twenty-eight days earlier loss of vision of both eyes within few days. Fundus negative. Twenty-four days, weakness of legs with Brown-Séquard syndrome progressing to complete loss of sensation, motion and tendon reflexes in legs	24 days	Died	Meningitis; hæmorrhagic, purulent central myelitis	Middle dorsal and lumbar	Streptococci; Gram-negative bacillus	Onset with retrobulbar neuritis. Diagnosis: meningitis, multiple myelitis
Hitchcock, 1917	M. adult	Infection, hand	Horsebite on hand with infection. Thirty-nine days before, progressive weakness of left leg, pain in left abdomen, dysuria; leucocytes, 12,000. Increased tendon reflexes. Twenty-six days right leg weak, reflexes almost absent. Twenty-five days both legs paralysed; pain-sensation absent below twelfth dorsal, tactile normal. Temperature of sepsis. Ascending paralysis to fourth dorsal. Sixteen days laminectomy; negative exploration but cord not incised. Turbid spinal fluid containing staphylococci	39 days	Died	Thick, creamy pus, lower 7 inches of cord; anterior horns involved in upper dorsal	From upper dorsal down	Staphylococci	..
Lesné and Belloir, 1922	M. 44	..	Tumours (actinomycosis) on thigh and shoulder. Five days before, loss of motion and sensation below umbilicus; incontinence; tendon reflexes absent; cerebro-spinal fluid 8 cells, trace albumin	5 days	Died	Limited to cord; meninges normal; central cord abscess	Lower dorsal	Actinomyces	..
Moersch, 1922	M. 25	Injury to eyelid	Cut right eyelid with whip. Seven months right ptosis; deafness; burning pain and numbness right leg; blurred fundi; decreased sensation right forehead; weakness of right leg; cerebro-spinal fluid. Nonne plus; 1,889 polymorphonuclears, 587 small lymphocytes; Lange 0111221000. Second day loss of patellar and tendo Achillis reflexes	7 months	Died	Actinomycotic cerebro-spinal meningitis; entire central canal filled with pus	Entire central canal	Actinomyces	..

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DECEREBRATE RIGIDITY OF THE SLOTH.[1]

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SHERRINGTON has shown that in quadrupeds like the cat, dog, guinea-pig and monkey, transection of the brain-stem near the level of the anterior colliculi produces a marked rigidity of all the muscles which normally assist the animal in maintaining its standing posture against the pull of gravity. These are chiefly the extensor muscles of the limbs and spine, the retractor muscles of the head and tail, and the elevator muscles of the jaw. An animal that has been decerebrated in this way stands without support when it is placed on its feet, and because of the dependence of this posture on proprioceptive impulses Sherrington termed it " reflex standing."

Our knowledge regarding this interesting phenomenon is limited almost entirely to observations made on quadrupeds which normally stand upright on all four feet. It occurred to one of us while studying the behaviour of the sloth, that this animal, because it has just the opposite posture and normally hangs rather than stands, would offer an excellent opportunity for establishing the general applicability of our present knowledge regarding decerebrate rigidity. Would decerebration in the sloth produce extensor rigidity as in the cat and dog, or flexor rigidity in keeping with its normal hanging posture? Would not the decerebrate sloth show " reflex hanging " rather than " reflex standing "? The results of our work answer these questions quite definitely, and at the same time suggest what posture one might expect in decerebrate man.

[1] The experimental part of this work was done by Dr. Richter, with the aid and suggestions of Dr. George B. Wislocki, in Panama at the Institute for Research in Tropical America. Dr. Richter wishes to express his indebtedness to the Institute for the assistance and facilities offered for carrying on this and other work during the summer months of 1924 and 1925. Thanks are due especially to Mr. James Zetek, custodian of the laboratory, and to Dr. Ignanzio Molino, assistant custodian, for their tireless efforts in helping to procure the necessary materials and animals. The histological work was done in the Neurological Laboratory of the Phipps Clinic by Dr. Bartemeier. The work both in Panama and in Baltimore was greatly aided by the interest and encouragement of Dr. Adolf Meyer.

The Normal Behaviour and Posture of the Sloth.

Since the sloth is very little known either through first-hand contact or the literature, we wish to give a short account of its normal behaviour and posture before presenting the results of our experiments. It is a strictly arboreal animal, living in the tropical rain forests of Central and South America. For most of its life it hangs back downward from the branches of trees, and its behaviour in this posture is characterized by marked inactivity, occasionally interrupted by slow and deliberate movements.

Fig. 1A.—Two-toed sloth hanging from lattice. This photograph shows it in an active posture, in contrast to the resting or sleeping posture in the next figure.

It is well equipped for this inverted hanging position with large strong claws from 6 to 10 centimetres long on all four legs. The action of these claws is entirely dependent on neuro-muscular activity, not on any mechanical arrangement of the tendons or ligaments which would enable the animal to hang with no expenditure of energy. This is demonstrated most clearly by the fact that the claws of the dead sloth are flaccid and toneless, quite unable to support the weight of the body.

In describing the details of the normal posture of this animal it is necessary to deal separately with the two common species, both of which were used in the following experiments. There is the *Cholœpus*

didactylus, Linn., species (see fig. 1 A), commonly called two-toed sloth from the number of claws on the fore-feet, and the three-toed *Bradypus griseus griseus* species (see fig. 2).

Both species have two normal postures, one assumed when resting or sleeping, the other when in motion or on the alert preparatory to moving. In the resting or sleeping postures of the two-toed sloth (see figs. 1 B and 1 C) the limbs and trunk are flexed, the head and tail are

FIG. 1B.—Two-toed sloth in sleeping posture. The head is tucked in between the fore-limbs, but is still just visible at the left end in the photograph. The hair was shaved from the right fore-limb. The bands around the fore-arm and the attached wires were used in some electromyographic experiments.

flexed ventrally. In this posture the head is almost completely concealed between the limbs, so that it becomes difficult to determine its location in the photograph. In the resting posture the animal appears as a large hairy ball. Its moving posture (see fig. 1A) is somewhat different. The hind-limbs, although still flexed, are less so than in the resting posture, and the fore-limbs are almost fully extended. The claws, of

FIG. 1C.—Two toed sloth. Same as 1B.

FIG. 1D.—Two-toed sloth in the prone position on a paved surface, unable to lift its weight from the ground.

course, are very markedly flexed. The head remains nearly in line with the axis of the body.

The helplessness of the animal on the ground is demonstrated in

FIG. 2.—Active hanging posture of three-toed sloth.

FIG. 3.—Three-toed sloth in its resting or sleeping posture, holding on to the trunk of a tree. It will be noticed that most of the work of holding is done by the hind-limbs which are elevated before the body. (Photographs by James Zetek.)

fig. 1D. It has become so highly specialized for its arboreal existence that when it does leave the trees on rare occasions it is scarcely able to raise its body from the ground and literally has to drag itself along.

The three-toed sloth normally exhibits resting and active postures which are very similar to those of the other species. In addition, however, it assumes a second resting posture when it sits at the top of a tree, clinging to the trunk (see fig. 3). We have described this attitude as "holding" rather than as "hanging," although the same muscles, the flexors, are involved in both. The hind-limbs, elevated at the thigh, spread apart, rotated outward, and flexed at the knee, form a plane nearly perpendicular to the longitudinal axis of the body. The depression of the head and the right-angle flexion of the elbow-joint further contribute to the general equilibrium, but the animal merely holds on to the tree with the claws of the fore-limbs, whereas it literally embraces the trunk with the hind-limbs. These are essential for the maintenance of the holding posture and anatomically well adapted to it, since they are short and stocky, and so strong that the animal searching for food can completely release the fore-limbs and lean backwards. The entire body is supported in this awkward position by the hind-limbs and the abdominal muscles.

Methods.

In decerebrating the sloths the technique used by Bazett and Penfield on cats was followed. In the sloth, however the complete operation could be performed by one man alone. After the animals were anæsthetized a mid-line incision was made in the skin of the neck and the common carotids exposed. A loop of silk thread passed carefully around each vessel was permitted to hang freely from the incision. Just before the transection of the brain-stem was performed, a small weight, a mosquito hæmostat or a similar object, was attached to the end of each loop and allowed to hang freely so that the flow of blood to the brain was temporarily stopped. Then the skull was trephined; an area of the brain about three-quarters of an inch in diameter was exposed just over the occipital region, and the plane was located through which the transection was to be made. During the operation the head was held in a horizontal position in the left hand, with the tips of the forefinger and thumb compressing the vertebral arteries between the wings of the atlas. The transection was effected by a single quick thrust through the hemispheres and brain-stem with a sharp scalpel. The severed part of the brain was immediately scooped out with the handle of the scalpel and the resulting cavity filled with cotton wool. The pressure on the vertebral arteries was then gradually released and the weights attached to the loops around the carotids

were removed. Because the slight bleeding in the animals stopped just as readily when no effort was made to prevent the flow of blood to the brain, the precautions for occluding the arteries were gradually dis-

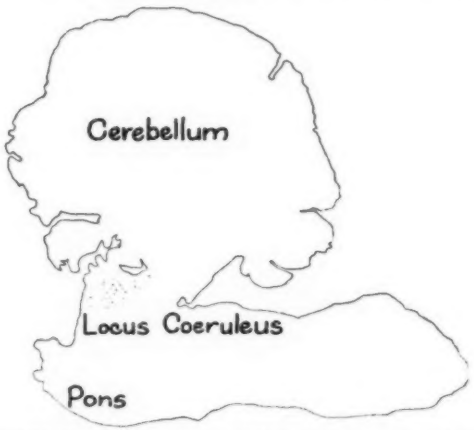

FIG. 4A.—Ink tracing of sagittal section, showing one of the brain-stems with the lowest transection. The section shows the cerebellum and indicates the position of the lower portion of the locus cœruleus; it is well below the red nuclei.

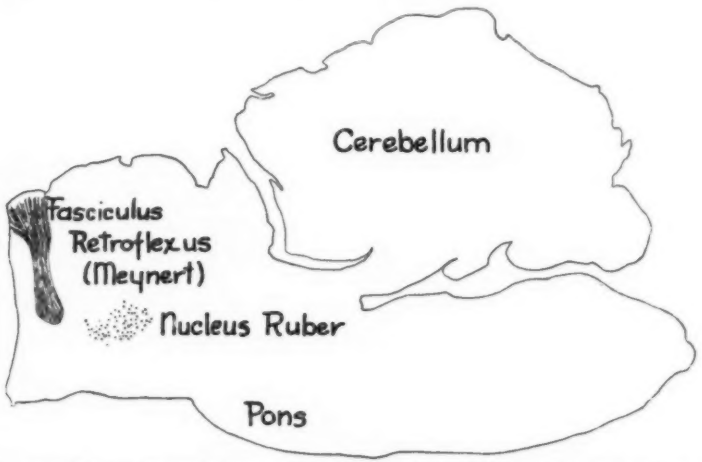

FIG. 4B.—Ink tracing made from section, showing level of highest cut made in any of the sixteen animals. The red nucleus and retroflex bundle of Meynert are indicated.

regarded. This greatly simplified the procedure, making unnecessary the incision in the skin of the neck and the search for the carotids. The entire operation was performed in two to three minutes, most of

the time being consumed in trephining the skull. Chloroform was used as an anæsthetic in three of the animals; ether, in the other thirteen, since ether was found to be far more satisfactory. Contrary

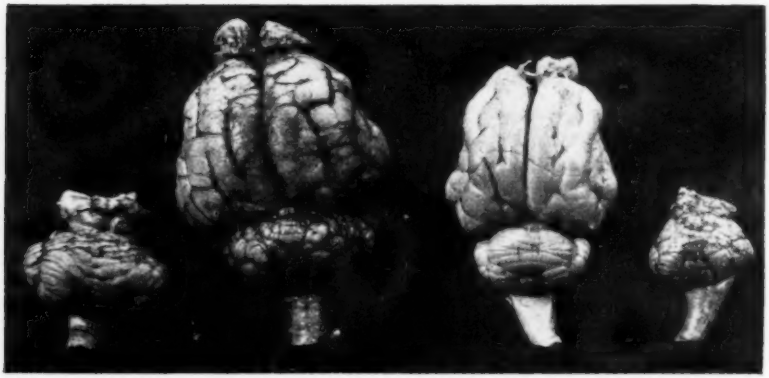

FIG. 5.—Photograph of an intact brain of a two-toed and of a three-toed sloth; also a brain-stem of a decerebrate individual of each species.

FIG. 6.—Two views of a decerebrate three-toed sloth (brain transection below red nuclei), showing flexion of limbs, depression of head, marked flexion of trunk. This posture is an almost perfect reproduction of the normal resting posture.

to current conceptions, it was found that the sloths became deeply anæsthetized very quickly, often in less than a minute.

Sixteen animals were decerebrated, three two-toed and thirteen three-toed sloths. The plane of transection varied between the lower level of

the locus cœruleus (see fig. 4A), in the upper hind-brain and the retroflex bundle of Meynert (fig. 4), in the anterior end of the mid-brain. We were fairly successful in our efforts to transect the brain-stem of each successive sloth at a slightly higher level. A normal and decerebrate brain of each of the two species is photographed in fig. 5.

From the work on other animals, especially cats, it is known that the success of keeping decerebrate preparations alive and in good condition depends very largely on how well the body temperature is maintained at or near the normal level. Since one of the most important characteristics of a decerebrate animal is its loss of temperature control, it is necessary to provide artificial heat regulation for decerebrate specimens. This is done either by heating the surrounding medium to the normal temperature, as was done by Bazett and Penfield [1], or, less elaborately, by using an electric pad or hot-water bottle and blankets. In Panama, where our sloths were decerebrated, the temperature of the air averaged about 85° F.; the normal body temperature of the animal is 85-91° F. The temperature of the air, then, served to maintain the body temperature of the sloths very near the normal level and greatly simplified the post-operative precautions. The excessive humidity of the tropics also aided in keeping the decerebrate animals in good condition.

Results.

In the sloth decerebration produces flexor rigidity of the limbs and trunk, and "reflex hanging" instead of extensor rigidity and "reflex standing" as in the decerebrate cat (see figs. 6, 7, 8, 9).

The intensity of the flexor rigidity and the ability of the animals to hang reflexly varied with the levels at which the brain-stem was transected, but especially with the position of the transection in relation to the red nuclei.

The five animals in which the red nuclei were entirely removed showed a very close reproduction of the sloth's normal resting or sleeping posture. Fig. 6 gives two views of one of the three-toed sloths in this group. The hind-limbs were held before the body, flexed at the knee and hip just as they are normally when encircling the trunk of a tree; the fore-limbs were flexed at the elbow and resisted forward movements at the shoulders; the trunk tended to become curled up and offered strong resistance to efforts to straighten it; the head and the short stubby tail were flexed ventrally; the claws also showed flexor rigidity. When the claws were hooked over a rod held horizontally, or

FIG. 7.—Photograph showing posture of three-toed sloth in which the brain-stem was transected above the red nuclei.

FIG. 8.—Reflex hanging posture of three-toed sloth decerebrated above red nuclei.

when the limbs were placed around the trunk of a small tree, the animal hung for several minutes without falling. The fore-limbs were invariably the first to let go, while the hind-limbs still maintained a fairly good grip. Thus it was shown that the animals without the red nuclei exhibited strong flexor rigidity of the extremities and trunk, closely reproducing normal resting or sleeping posture, but because of the poor tonus of the claws they were not able to maintain their normal hanging posture very long before they fell.

The eleven sloths with the transection either through or above the red nuclei showed less rigidity, especially of the fore-limbs and trunk,

FIG. 9.—Reflex hanging posture of two decerebrated two-toed sloths. The level of transection was above the red nuclei in both. The animal at the right remained hanging for over thirty hours after the operation. The hair was clipped from the limbs to show their posture.

but a much more marked rigidity of the claws, so that they were able to hang much more steadily and longer than the animals in the first group. One of the three-toed sloths with the high transection is seen lying on its back in fig. 7. The fore-arms are flexed at the elbows; the hind-limbs are flexed at the knee and show strong resistance to being spread apart, but are not drawn up before the body as they are in the other sloths; the head is bent neither forward nor backward, but tends to remain in line with the axis of the body. When hanging, these animals very closely reproduced the sloth's normal active posture. (See the photograph of the decerebrated three- and two-toed sloths in figs. 8 and 9.) The fore-limbs are almost fully extended, although before the

animals were placed in the hanging position they were flexed; the hind-limbs are flexed, but not so much as in the resting posture; the head is held in good position. Even when hanging by only two limbs, as in fig. 10, the animals were able to maintain their hanging posture very well. Thus it was demonstrated that these animals with the higher transection show less rigidity of the limbs and trunks but more of the claws, so that they are much better able to maintain their normal hanging posture.

FIG. 10.—Two-toed sloth decerebrated above the red nuclei. It is shown hanging from only two legs

One animal remained hanging for over thirty hours after the operation. After that time the grip of its fore-limbs gave way, while that of the claws of the hind-limbs was still as strong as before.

These results demonstrate that to a large extent the rigidity of the limbs and trunk is distinct from the rigidity of the claws upon which "reflex hanging" depends. The animals in which the red nuclei were removed showed strong rigidity of the limbs and trunks, but were scarcely able to hang, while those in which the nuclei were left intact showed much less marked rigidity of the limbs and trunk but were

able to hang very well for long periods of time without showing any fatigue.

This observation is essentially in agreement with the results of Magnus and Rademaker [2] [3], in their experiments on the red nuclei and their relation to postural tone. They found that cats decerebrated below the red nuclei show a marked rigidity of the limbs and trunk. In this respect they are like the sloths with similar transection. Magnus and Rademaker showed, however, that the standing posture of the decerebrated cats is only a caricature of the standing posture of a normal animal, because of the exaggerated tone in the extensors and the almost complete absence of tonus in the flexors. From some of our own observations made on thirty cats we have concluded that much of the discussion regarding the exaggerated nature of the standing posture of decerebrated cats is overdrawn. It is true that when these animals are examined lying on their sides they show a very strong extensor rigidity of the limbs, but when they are placed in the standing position they can maintain it for a few minutes only. The same was true of the sloths without the red nuclei; in spite of the marked rigidity of their limbs they were able to hang but a few minutes.

It seems likely that the failure to distinguish between rigidity of the limbs and ability to stand may explain some of the differences in experimental results obtained by different workers.

Furthermore, Magnus and Rademaker found that animals in which the red nuclei were left intact showed less rigidity but a normal distribution of tone and a normal standing posture. The sloths with the transection above the red nuclei also showed less rigidity but were able to hang much longer than the animals of the other group.

Reproduction of normal posture in the decerebrate sloth is shown further by the order in which the different parts of the total posture appear and disappear after operation. It was pointed out above that in the normal individual the hind-limbs play the most important rôle, since by them alone the animal is able to maintain its position. It is an interesting fact, then, that in the decerebrate sloth the hind-limbs are the first to become flexed and rigid and the last to lose their rigidity. They usually show good flexor tonus almost immediately after the brain-stem is transected. The flexion of the claws of the hind-feet appears about the same time. Whether the knee or hip becomes rigid first we were not able to determine. Usually soon after decerebration the hind-limbs, showing right-angle flexion, are held as if fastened around a tree, and offer resistance to spreading. Flexor tone of the

claws on the fore-limbs, relatively less essential for the normal animal to preserve its equilibrium, appears next. Then the elbows show rigidity. We were unable to discover when the tonic contractions of the trunk muscles appeared.

Thus it is shown that the most important effects of decerebration appear in the different parts of the animal in the order of their importance for the maintenance of the normal posture. It is of interest to recall here that the decerebrate cat, which normally uses its fore-limbs

FIG. 11.—Photograph of two-toed sloth, showing well-directed response of fore-limbs to slight touch on incision at back of head. This animal often actually succeeded in tearing the sutures and in pulling out cotton swabs from the incision.

most in preserving its erect posture, shows extensor rigidity in the fore-legs earlier and to a greater degree than in the hind-legs.

Further light is thrown on the relation between the characteristic attitude of the normal and decerebrate sloths by postural differences existing between the two species and concerning chiefly the hind-limbs. In the three-toed sloth, as has been pointed out, the hind-limbs may be held up before the body with the thigh elevated and the leg abducted and rotated outward. In the two-toed sloth the hind-limbs, which are much longer than those of the three-toed species, are used more

distinctly for hanging than for holding, so that they show neither spreading nor rotation. This difference is present likewise in the decerebrate animals.

The flexor rigidity of the sloths exhibits, in common with extensor rigidity, inhibition due to phasic reflexes. Almost any tactual stimulation around the head will cause the flexor tonus of the fore-limbs to disappear in favour of an extending and clasping movement, often directed with marked accuracy to the point of stimulation (see fig. 11), or as though it were attempting to catch the offending object in its arms.

It may be pointed out in this connection that a study of the phasic reflexes and their resulting effect on the flexor rigidity disclosed, quite by chance, the fact that these animals react also to at least one acoustic stimulus. We had previously made repeated vain attempts to elicit responses by using all kinds of instruments for producing noises. On one occasion, however, when a normal baby sloth was examined in the presence of a decerebrate adult sloth, it was found that every time the baby squealed the adult raised its body off the table and turned its head slowly from side to side. This observation was repeated many times always with the same result. It was a noteworthy fact that a response could be obtained by this one acoustic stimulus and no other.

Although phasic reflexes affect the fore-limbs very readily, it is an interesting fact, in keeping with the account given above regarding the importance of the hind-limbs in the posture of the animal, that these reflexes have very little, if any, effect on the hind-limbs. Were their flexor tone to become inhibited and the hind-limbs relaxed under normal conditions, the animals would fall to the ground.

The lengthening and shortening responses characteristic of extensor rigidity are present with some modifications in flexor rigidity. This may be demonstrated by an examination of the rigidity of the fore-limb. We start with the limb in a fully flexed position and extend it slowly. The extension movement meets with almost no resistance until the fore-arm reaches a position in which it is nearly perpendicular to the upper arm. Then suddenly, as though a string from the shoulder to the wrist had become taut, marked rigidity appears; and from then on the usual lengthening response is present. This result seems to be partly in disagreement with the recent findings of Liddell and Sherrington [4] on the so-called myotatic or muscle-stretch reflexes; they have shown that even the slightest pull on an extensor muscle of a decerebrate cat is sufficient to produce a reflex contraction of the

muscle, and they explain the phenomenon as follows. The stretching of the muscle stimulates sensory endings within itself, which set up impulses that are carried from this muscle to the cord and then back again to the muscle. In the sloth, however, as has just been shown, the fully flexed fore-arm can be extended until the upper and lower parts are nearly at right angles to each other before the rigidity begins. This means that during this time the biceps muscle is being subjected to a considerable amount of stretching without eliciting the reflex contractions which would be expected on the basis of Liddell's and Sherrington's work. It is not clear at present how these results can be explained. We may consider in this connection the fact that the right-angle position of the fore-arm and upper-arm occurs most frequently in the normal animal and affords the greatest mechanical efficiency.

Discussion.

These experiments clearly establish the general applicability of the conclusions regarding decerebrate rigidity arrived at by Sherrington. He has shown that decerebrate rigidity is a postural reflex involving the muscles used by the animal in maintaining its normal posture against the pull of gravity. The decerebrate sloth, just as one would expect on the basis of Sherrington's conclusions, shows "reflex hanging" with flexor rigidity of the limbs, trunk, and especially the claws. To explain this phenomenon, however, simply by saying that the muscles showing rigidity are anti-gravity muscles, gives only a part of the picture. It does not explain how they are affected. The limitations of this interpretation come out much more clearly in the sloth with its general state of flexion than in animals with extended limbs and body. It is true that in the decerebrate sloth the anti-gravity muscles, the flexors, are involved, but this does not explain why the fore-limbs, for instance, happen to be flexed almost exactly to a right angle. One might have expected to see them fully flexed, just as the limbs of a cat are fully extended. It does not explain why the hind-limbs of a decerebrate sloth without the red nuclei are also held in a position of semi-flexion, with the thighs elevated at the hip until they are nearly at right angles to the axis of the body, rotated outward and spread, with the right angle flexion at the knee-joint. An understanding of the origin of these special postures is obtained when we learn that they reproduce in almost every detail the animal's habitual posture. Sherrington somewhat indirectly has also called attention to this point. We should like to emphasize further its significance.

On the basis of our present knowledge regarding decerebrate rigidity, derived on the one hand from animals which use their extremities for standing, and on the other from those which use them for hanging or for holding, it seems possible to foretell what kind of posture would be produced by a similar operation in man. His normal posture has, in common with that of standing animals, the extensor rigidity of the legs used in supporting the body, and with that of hanging animals, the flexor rigidity of the arms and hands used in lifting, holding, and carrying activities. Then, following the principle of the close reproduction of habitual posture by decerebrates, it seems reasonably certain that in man this operation would produce extensor rigidity of the legs, and flexor rigidity of the arms and hands.

This conclusion is of some importance for the study of conditions of decerebrate rigidity produced in man by lesions at different locations. Kinnier Wilson [5] described a number of cases which he believed showed the same features present in decerebrate rigidity produced experimentally in animals. For him this meant chiefly extensor rigidity of all extremities, as it is found in the decerebrate cat or dog.

Walshe [6] [7] thinks that Wilson's cases, with possibly one or two exceptions, do not show true decerebrate rigidity, but that they are instances of generalized muscular spasms such as are seen in tetanus and strychnine tonic fits and similar conditions.

In Walshe's opinion, true experimental decerebrate rigidity must show, besides Magnus-de Kleijn reflexes, reflex inhibition due to phasic reflexes, shortening and lengthening responses, extensor rigidity of the legs, and *flexor* (not extensor) rigidity of the arms and hands.

We were so fortunate as to be able to see a case of hemiplegia through the kindness of Dr. Frank R. Ford, of the Neurological Department, affecting chiefly the upper-arm, examined by Walshe during his stay at the Johns Hopkins Hospital in 1925. It was a paralysis of long standing and was described by him as showing all of the physiological features of typical experimental decerebrate rigidity. Our examination showed the presence of marked flexor rigidity of the forearm and the lengthening and shortening reaction, Magnus-de Kleijn reflexes, and the reflex inhibition due to phasic reflexes. But what from our point of view was especially interesting was that this patient showed the 90-degree flexion of the elbow-joint and the grasping reflex. An examination of the arm showed that, just as in the sloth, the rigidity began suddenly when the lower arm was nearly at right angles to the upper arm. Voluntary movement of the fingers was almost entirely

lost, but reflex grasping was present just as it is in the new-born infant and in the decerebrate sloth. A rod placed in the hand of the patient caused the fingers to contract around it so firmly that strong effort was required to detach it again.

These experiments definitely explain the appearance of flexor instead of extensor rigidity in the arms of man, showing true decerebrate rigidity, and clearly bear out Walshe's conclusions arrived at from a somewhat different angle.

This principle of habitual posture may throw some light on the differences in the position of the fore-limbs obtained by Sherrington and Riddoch in their work on monkeys. In some animals the fore-limbs were extended and in some way they were flexed. It would be important to know whether the kind of monkeys used in their experiments normally employed their fore-limbs as props for supporting the body weight, or for holding and gripping. The *rhesus* monkeys use their fore-limbs as props, while monkeys of some other species—for instance, the spider monkey, which we have had under observation for several months—do most of their walking with their hind-limbs and restrict the use of their fore-limbs to gripping and manipulating. It may be pointed out here that it is possible also that the differences in the habits of the individuals of each species may show up after decerebration.

Summary.

(1) The decerebrate sloth shows flexor rigidity of the limbs, claws and trunk, and reflex hanging in keeping with its normal posture. This is to be expected on the basis of the principle arrived at by Sherrington in his work on quadrupeds with an upright standing posture.

(2) The posture of the decerebrate sloth varies with the level of transection. When the red nuclei are removed the posture closely reproduces the animal's resting posture. In these animals the rigidity is marked in the trunk and limbs, but is not so strong in the claws. For this reason they are not able to hang very well. When the cut is made above the red nuclei, the rigidity of the limbs and trunk is less marked, but the ability to hang is much greater. The hanging posture closely corresponds to the animal's normal active posture. The agreement of this work with that of Magnus and Rademaker is pointed out.

(3) Similarities in the physiological characteristics of flexor and extensor rigidity are discussed. They show in common the lengthening

and shortening responses, reflex inhibition from phasic reflexes and lack of fatigue.

(4) The dissimilarities have to do chiefly with spastic quality. For a certain range of movement the limbs do not exhibit any rigidity. In the fore-limbs the rigidity does not manifest itself until the lower part of the limb is nearly at right angles to the upper part; then it appears very suddenly and in full strength. This is discussed at length and is referred to as the right-angle flexor phenomenon.

(5) It is pointed out that the posture of the decerebrate sloth cannot be completely explained by stating that the anti-gravity muscles were involved. It is necessary to refer also to the habitual posture of the animal.

(6) On the basis of this principle it is shown what posture might be produced by a similar operation in man. Since in normal man the legs are extended in supporting the body, and the arms and hands flexed in lifting and holding, decerebate man would probably show extension of the legs and flexion of the arms and fingers.

An example is given of a case of hemiplegia which showed all of the criteria of true experimental decerebrate rigidity, Magnus-de Kleijn reflexes, lengthening and shortening responses, and reflex inhibition due to phasic reflexes. In addition, it showed extensor rigidity of the legs and flexor rigidity of the arms, thus confirming Walshe's conclusions arrived at from a somewhat different angle.

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THE COLLOIDAL PARAFFIN REACTION IN THE CEREBRO-SPINAL FLUID.

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INTRODUCTION.

COLLOIDAL reactions in the cerebro-spinal fluid are of undoubted value in diagnosis, and, although several have been described, that originated by Lange is the only one which has become a routine investigation. Its chief advantage lies in its delicacy in detecting physico-chemical abnormalities in the fluid in organic nervous disease when other tests, serological or purely chemical in nature, prove negative. This paper is a record of the examination of 130 cerebro-spinal fluids by a test in which the colloidal reagent is a suspension of paraffin wax in a water-alcohol solution—a colloid which is easy to prepare and is cheap. This reagent and its application to cerebro-spinal fluid examination was first described by Kafka in 1925. It is a sensitive indicator, and from the results which follow we consider it to be superior to the Lange reagent, which is costly, unstable, and difficult to prepare. In order to discover the best technique for the colloidal paraffin test I have experimented with Kafka's actual method, and with two modifications which I have called "B" and "C." The majority of tests were made using modification "B."

The cerebro-spinal fluids were derived from patients at the National Hospital, Queen Square, London, the Bristol General Hospital, the Southmead Hospital and the Bristol Mental Hospital, and I wish to express my indebtedness to the staffs of these hospitals for their kindness in allowing me to examine these specimens.

The cases were mostly those of chronic nervous diseases, although several cases were included in which no evidence of organic disease could be detected. Such instances included the neuroses, psycho-neuroses and epilepsy.

TECHNIQUE.

Modification "B" is described first, as it was the method most employed in these tests.

Colloidal Paraffin B.

(1) A *stock paraffin solution* is prepared from a block of Grübler's white paraffin wax, with a melting point of 52° C. This is cut by a microtome into flakes, each of 5 microns in thickness, and 0·25 gm. is weighed out. This wax is then dissolved in 100 c.c. of absolute ethyl alcohol, at a temperature of 52° C. On cooling a flocculent suspension results, which clears on heating to a temperature just below that of the melting point of the wax. This constitutes the stock solution and can be kept for many weeks.

In order to prepare the *working solution* the stock solution is warmed to 52° C.; 10 c.c. are taken, to which are added quickly 30 c.c. of twice distilled water, warmed to the same temperature. An opalescent colloidal suspension results which remains unchanged on cooling. This suspension must be prepared freshly for every batch of fluids.

(2) A 0·2 *per cent. sodium chloride solution* is made with twice distilled water.

(3) Eleven test tubes, each 3½ in. by ½ in., are employed for this test. They must be of good resistance glass; scrupulous cleanliness must be ensured by washing first in sulphuric acid and potassium bichromate, then in successive washings of distilled water. They are dried in a hot-air oven.

(4) *The actual test*: 0·5 c.c. of the saline is measured into each tube, followed by 0·75 c.c. of the working paraffin solution. Into the first tube 0·25 c.c. of cerebro-spinal fluid are measured, 0·5 c.c. are transferred from this tube to tube 2 and the contents mixed, 0·5 c.c. of the mixture are transferred to tube 3, and so on as far as tube 10, from which 0·5 c.c. are discarded. Tube 11 serves as a control, containing only paraffin and saline.

The tubes are left at ordinary room temperature for twelve hours before reading the results.

The dilutions of cerebro-spinal fluid in the tubes are 1 in 6 : 35, &c.

As in the other colloidal tests the various degrees of precipitation are indicated arbitrarily by numerals:—

No change	=0
Slight precipitation of wax with no clearing of the fluid	=1
Slight precipitation of wax with slight clearing of the fluid	=2
Heavy precipitation of wax with considerable clearing of the fluid	=3
Heavy precipitation of wax with complete clearing of the fluid	=4

Thus full precipitation, indicated by the numeral "4" in the paraffin tests, corresponds with the numeral "5" in the Lange reaction.

Specimens of cerebro-spinal fluid contaminated with blood are

discarded, because the serum globulins are known to cause precipitation. The fluids are tested as soon as possible after withdrawal, although there is no evidence that different results are produced even after a week's exposure at room temperature, provided the specimen was sterile on collection and remains so. Fluids from cases of tuberculous and pyogenic meningitis, and from acute anterior poliomyelitis should be examined within twenty-four to thirty-six hours. Whenever a fresh stock solution of paraffin is made up it is advisable to test its sensibility against known normal and paretic fluids.

Kafka's Technique.

Colloidal Paraffin A.

The stock solution of paraffin is 0·3 per cent.

The working paraffin solution contains equal parts of stock paraffin solution and twice distilled water. The NaCl solution is 0·3 per cent.

Test.—Into each of eleven tubes put 0·5 c.c. NaCl solution.

Into tube 1 put 0·5 c.c. cerebro-spinal fluid. Mix and transfer 0·5 c.c. into tube 2, and so on, discarding 0·5 c.c. from tube 10.

Into each tube put 1 c.c. of the working paraffin solution. Mix.

Thus the dilutions of the cerebro-spinal fluid in this test are :—1 in 2 : 4 : 8 : 16 : 32 : 64 : 128 : 256 : 512 : 1024 :

Colloidal Paraffin C.

The stock paraffin solution is 0·2 per cent.

The working solution is one part of stock paraffin solution in three parts of water.

The saline solution is 0·2 per cent.

Test : Into each tube put 0·5 c.c. NaCl solution.

Into tube 1 put 0·25 cerebro-spinal fluid. Mix and transfer 0·5 c.c. into tube 2 and so on.

Into each tube put 0·75 c.c. of colloidal paraffin. Mix.

The dilutions of the cerebro-spinal fluid in this modification B are :—

1 in 4 : 8 : 16 : 32 : 64 : 128 : 256 : 512 : 1024 : 2048.

Although, as I have pointed out, none of the patients can be regarded as "normal" in the strict sense, nevertheless the absence of physical evidence of organic disease, together with a completely normal cerebro-spinal fluid, constitute the nearest approach to normality that has been possible. The results of the paraffin reaction may therefore be considered from various clinical points of view.

(*A*) *Normal subjects*, i.e., cases without physical evidence of organic disease and with normal cerebro-spinal fluids (neurosis, psycho-neurosis, &c.). 9 cases.

(*B*) *Normal cerebro-spinal fluids obtained from cases of organic diseases.* 52 cases.

(*C*) *Pathological cerebro-spinal fluids.* 70 cases.

Class A.

Nine patients diagnosed as suffering from functional nervous disorders yielded cerebro-spinal fluids which were quite normal with respect to cytology, protein content, Wassermann and Lange tests. In six of the patients, no precipitation of wax was seen in the colloidal paraffin test. In three of them, a very slight degree of precipitation occurred in one or two tubes containing the lower dilutions, never exceeding the figure "1." As with the other colloidal tests, this slight precipitation in low dilutions must be regarded as of no significance.

Class B.

Fluids were examined from numerous neurological patients, all of which gave normal cytological, protein, Wassermann and Lange tests. In such fluids the colloidal paraffin test was either uniformly negative, or else gave a degree of precipitation not exceeding "2."

Epilepsy (6 cases). Negative paraffin curves were obtained in five, in one case only the curve 0012100000 was obtained; in this case, four cells were counted in 1 c.mm., the total percentage of protein was 0·025, there was no excess of globulin, the Wassermann was negative, and the gold-sol curve read 0001000000.

Cerebral tumour (10 cases). In every case the paraffin curve was negative.

Cerebellar tumour (6 cases). Five cases gave completely negative readings, whilst in the sixth the curve 0011210000 was obtained, although all the other reactions, including the colloidal gold, were negative.

Miscellaneous diseases.

Aortic regurgitation (1 case).
Cerebral abscess (2 cases).
Chronic uræmia (1 case).
Hæmatomyelia (2 cases).
Hemiplegia (3 cases).
Huntington's chorea (1 case).

Familial spastic diplegia (1 case).
Mumps (1 case).
Olivo-ponto-cerebellar atrophy (1 case).
Old cerebral hæmorrhage (1 case).
Paraplegia (1 case).
Pituitary tumour (2 cases).
Post-encephalitic Parkinsonism (1 case).
Progressive muscular atrophy (3 cases).
Secondary malignant growth in spine (2 cases).
Sciatica (1 case).
Spastic paraplegia (1 case).
Subacute combined degeneration (1 case).
Tetanus (2 cases).
Undiagnosed (3 cases).

Most of these cases yielded absolutely negative paraffin curves, associated with complete negativity in the other tests.

In one case of infantile hemiplegia, where the fluid showed a slight globulin increase, but a negative Lange test, the paraffin curve was 0121000000.

One case, diagnosed as olivo-ponto-cerebellar atrophy, gave a curve 0121111100; there was some increase in globulin, but there was no precipitation in the Lange test. One case of sciatica gave a paraffin curve 0122110000 in an otherwise normal fluid.

Class C.

The pathological fluids examined fall mainly into four classes:—
(1) Neuro-syphilitic disease.
(2) Disseminated sclerosis.
(3) Acute infections of the central nervous system.
(4) Fluids showing a partial or complete loculation syndrome.

C (1). *Neuro-Syphilis.*

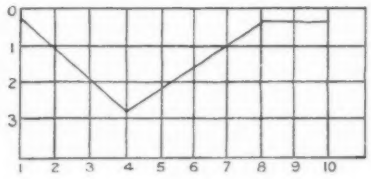
Case No.	Age	Sex	No. of cells	Total protein %	Nonne-Apelt	Pandy	Wassermann	Lange	Colloidal paraffin A	Colloidal paraffin B	Colloidal paraffin C
(A) Congenital (two cases): (1) Congenital G.P.I.; (2) Optic atrophy.											
1	17	Female	80	0·1	++	++	C.S.F. 4444 Blood 4444	3542333210	..	4444210000	..
2	..	Female	3	0·06	+	++	Negative ..	0001110000	..	0000000000	..

Case No.	Age	Sex	No. of cells	Total protein %	Nonne-Apelt	Pandy	Wassermann	Lange	Colloidal paraffin A	Colloidal paraffin B	Colloidal paraffin C
	(B) Tabes (eighteen cases).										
1	61	Male	11	0·075	++	+	C.S.F. 4444 Blood 4320	0011122210	..	0112110000	..
2	46	Male	100	?	?	+	C.S.F. ? Blood 2100	0123221100	..	4432110000	..
3	48	Male	4	0·045	Weak +	Weak +	C.S.F. 4444 Blood 4444	0001111000	..	0011210000	..
4	40	Female	150	0·07	+	+	C.S.F. 4444 Blood 3200	0111253310	..	0011111000	..
5	48	Male	48	0·045	+	+	C.S.F. 4310 Blood 2000	0001123221	..	0002110110	..
6	..	Female	88	0·3	+++	+++	C.S.F. 4444 Blood 4420	0001122332	..	0011233210	..
7	45	Male	..	0·03	++	..	C.S.F. 4420 Blood 4444	0011234432	..	0110000000	..
8	..	Male	60	0·075	++	++	C.S.F. 4444 Blood 4310	0012212100	..	1211100000	..
9	..	Male	5	0·025	Neg.	Neg.	C.S.F. + Blood —	0001200000	..	1244432210	..
10	..	Female	27	0·05	++	++	C.S.F. 4210 Blood 2110	0001112100	..	0001211000	..
11	37	Male	1	0·04	+ —	Neg.	C.S.F. neg. Blood 1000	0001210000	..	0121110000	..
12	..	Male	2	0·02	Neg.	Neg.	C.S.F. neg. Blood 3210	0000000000	..	..	1112211100
13	..	Female	1	0·025	Neg.	Weak +	Negative ..	0000000000	..	..	0112111100
14	..	Female	0	0·05	Weak +	Weak +	C.S.F. 1000 Blood 1000	0011122110	0122100000	..	0012110000
15	..	Male	3	0·08	++	++	C.S.F. 4421 Blood 2210	0112233321	0123321000	..	0012110000
16	..	Female	3	0·035	Neg.	Neg.	C.S.F. 1000 Blood 2100	0000000000	0001100000	..	0000000000
17	..	Female	4	0·02	Neg.	Neg.	Negative ..	0111001000	1000000000	..	0000000000
18	..	Male	0	0·02	Neg.	Neg.	Negative ..	0000000000	0000000010	0110000000	0000000011
	(C) G.P.I. (thirteen cases).										
1	30	Male	130	?	?	++	C.S.F. ? Blood 4444	4554532110	..	3443210000	..
2	45	Male	30	0·075	++	++	C.S.F. 4444 Blood —	0111233211	..	4443211000	..

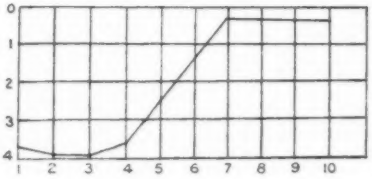
Case No.	Age	Sex	No. of cells	Total protein %	Nonne-Apelt	Pandy	Wassermann	Lange	Colloidal paraffin A	Colloidal paraffin B	Colloidal paraffin C
3	..	Male	15	0·045	+	..	C.S.F. 4444 Blood 4432	1323332110	..	1121100000	..
4	34	Female	..	..	..	..	C.S.F. — Blood+++	..	..	4442100000	..
5	42	Female	..	..	..	..	C.S.F.+++ Blood+++	..	..	3432100000	..
6	48	Male	20	0·06	++	++	C.S.F. 4444 Blood 4420	2455443321	..	4431000000	..
7	..	Male	45	0·06	+	+	C.S.F. 4444 Blood 4443	3443553221	..	3442100000	..
8	..	Male	25	0·065	+	+	C.S.F. 4431 Blood —	2333221111	..	0112110000	..
9	50	Male	70	0·06	+	+	C.F.S. 4444 Blood 4443	4554543110	..	3210000000	..
10	..	Male	35	0·08	++	++	C.S.F. 4442 Blood 4310	4455532100	1444310000	2432100000	3431000000
11	..	Male	87	0 01	++	++	C.S.F. 4444 Blood 4421	4555554210	2444431100	4442100000	3442000000
12	..	Male	..	....	..	..	..	..	..	..	4443321110
13	..	Female	24	0·045	+	Weak +	C.S.F. 4443 Blood 4444	4455554322	..	..	4444321110
	C (2).					*Disseminated Sclerosis.*					
1	..	Female	3	0·025	Neg.	Neg.	Neg.	0011121000	..	0011111000	..
2	37	Male	0	0·03	Neg.	Very weak +	Neg.	0001000000	..	0010000000	..
3	..	Male	0	0·03	Neg.	Neg.	Neg.	0000000000	..	0010000000	..
4	..	Male	0	0·05	Very weak +	+	Neg.	0001122100	..	0012111100	..
5	..	Male	0	0·03	Neg.	Neg.	Neg.	0023330000	..	0000000000	..
6	38	Male	0	0·02	Neg.	Neg.	Neg.	0000220000	..	0000010000	..
7	30	Male	4	0·03	Neg.	Neg.	Neg.	0012322110	..	3321000000	..
8	46	Female	5	0·045	+	+	Neg.	0011211000	..	0000000000	..
9	..	Male	14	0·03	Neg.	Neg.	Neg.	3555554210	..	2344332110	..
10	..	Female	18	0·025	Neg.	Neg.	Neg.	0000000000	..	0000000000	..
11	..	Male	..	0·025	..	..	Neg.	0000000000	..	0001112110	..
12	4	Female	2	0·03	Neg.	Neg.	Neg.	..	..	0000000000	..
13	..	Female	5	..	++	++	Neg.	0001011234	1000000000	..	0000000000

NEURO-SYPHILIS.

The results obtained show that there is obviously a very close parallelism between the Lange and paraffin reactions which obtains for all the types of neuro-syphilis examined. As with the gold test, the curve typical of tabes is 0123211000, that is no full precipitation, the



TYPICAL TABETIC CURVE.



TYPICAL PARETIC CURVE.

highest figure being "3" and the peak of the curve being reached in the fourth tube. The paraffin curve typical of paretic fluids is characterized by complete precipitation in the first three or four tubes, the remaining dilutions rapidly becoming normal, thus: 4444321000.

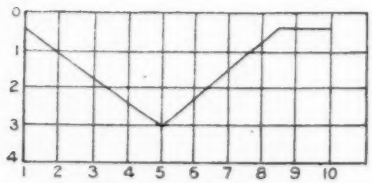
C (2). DISSEMINATED SCLEROSIS.

Of the thirteen cerebro-spinal fluids examined, four showed abnormal results. Two of these (Cases 7 and 9) gave curves of the paretic type. Two other fluids (Cases 4 and 11) presented luetic curves. These positive curves with the paraffin test corresponded with the results of the Lange test, which was performed in all except in one case, thus the paraffin reagent is shown to be very sensitive. In Case 13 the Lange gave precipitation in the last five tubes, but no abnormal results were obtained with the colloidal paraffin tests.

A paretic curve with one of the colloidal reactions and a negative Wassermann reaction is considered almost pathognomonic of disseminated sclerosis. Thus a fluid derived from a case of disseminated sclerosis which shows a normal total protein content and minimal chemical or cytological change, may still by the colloidal paraffin reaction show noteworthy abnormality.

C (3). *Meningitis.*

No. of case	Age	Sex	No. of cells	Type of cells %	Total protein %	Organism	Wassermann	Colloidal paraffin B
1	29	Male	200	90 lympho. ..	0·3	T.B. present	Negative	0123211000
2	5	Female	300	98 lympho. ..	0·3	None found	,,	0111122221
3	14	Male	400	98 lympho. ..	0·5	None found	,,	0011121110
4	8	Male	200	98 lympho. ..	0·1	None found	,,	0012321000
5	17	Female	2	..	<0·02	None found	,,	0000000000
	1st examination							
6	17	Female	..	..	..	T.B. present	,,	0000000000
	2nd examination							
7	17/12	Male	200	90 lympho. ..	0·05	T.B. present	,,	0000000000
8	1	Male	200	50 lympho. .. 50 leuco.	0·1	None found	,,	0011113210
9	11	Male	..	..	..	Staphylococci	,,	0012110000
10	..	Male	145	..	0·15	None found	,,	3211110000
11	4/12	Male	130	90 lympho. ..	0·15	None found	,,	3321100000
12	..	Male	340	85 polymorph.	0·15	Cholestrine	,,	0000000000



TYPICAL MENINGITIC CURVE.

(*a*) *Meningitis.*

Tuberculous meningitis gives abnormal curves. On an average the peak of the curve given by such a fluid was in the fifth tube, which showed marked precipitation of wax with considerable clearing of the solution.

The meningitic curve differs from the tabetic by having its crest at the fifth tube instead of the fourth. In two cases (Cases 10 and 11 a curve resembling that of general paralysis in conformation, but differing in its incomplete precipitation, was obtained.

(*b*) *Acute Anterior Poliomyelitis.* (Two cases.)

Case No.	Age	Sex	Day of disease	No. of cells	Total protein %	Nonne-Apelt	Pandy	Wassermann	Lange	Colloidal paraffin B
1	19	Male	10	80	0·045	Neg.	Very weakly +	Neg.	0001100000	0122111100
2	16	Male	9	14	0·1	++	++	Neg.	0001121210	0111110000

The only two cases of acute poliomyelitis tested gave slightly abnormal curves, but the precipitation was not so marked as with neuro-syphilis or meningitis.

(*c*) *Peripheral Neuritis.* (One case.)

Case No.	Age	Sex	No. of cells	Total protein %	Nonne-Appelt	Pandy	Wassermann	Lange	Colloidal paraffin B
1	64	Male	1	0·06	Neg.	Neg.	Neg.	0011221100	0122110000

C (4). *Fluids showing Partial Loculation Syndrome.*

No example of complete Froin's syndrome was examined, but in several cases a mild degree of protein increase indicated a probable compression of the spinal cord.

Case No.	Age	Sex	No. of cells	Total protein %	Nonne-Apelt	Pandy	Wassermann	Lange	Colloidal paraffin B
Spinal tumour.									
1	..	Male	6	0·06	Very weak +	Very weak +	Neg.	0000000000	0000000000
2	24	Male	19	0·05	Very weak +	Very weak +	Neg.	0011221100	0000000000
3	..	Male	12	0·07	Very weak +	Neg.	Neg.	1112211000	0110000000
Root lesion, L 2. 3. 4.									
1	..	Male	1	0·035	Very weak +	Very weak +	Neg.	0000000000	0000110000
Root lesions, S 4. 5.									
1	37	Male	1	0·04	+ −	Neg.	C.F.S. neg. Blood 1000	0001210000	0121110000
Lesion of cauda equina.									
1	..	Male	0	0·03	Very weak +	Very weak +	Neg.	0001100000	0121000000
Von Recklinghausen's disease.									
1	..	Male	0	0·06	Very weak +	Very weak +	Neg.	0000001110	2111100000
2	22	Male	2	0·075	+	+	Neg.	0001121100	1110000000

From the results of the cases recorded, it will be observed that there is a rise of total protein, and that the protein chiefly affected in this increase is the albumin. These cases gave either negative or only mildly positive curves with the colloidal tests, such peculiar readings being common in fluids of this nature. Felton and Cruickshank have found that it is the globulin which causes the precipitation of the colloidal solutions; it is probable, therefore, that in Froin's syndrome the effect of the increased globulin is masked by the protective action of the albumin.

A brief comparison of the results of the colloidal paraffin reaction with those of the routine tests is of interest.

Total protein.—All fluids with a total protein over 0·05 per cent., with the exception of the cases of spinal compression, gave abnormal curves. The highest protein percentages were obtained in tuberculous meningitis, but they did not produce total precipitation, whilst paretic fluids with less protein caused total precipitation. Three cases of disseminated sclerosis with protein contents of less than 0·045 per cent. gave abnormal curves. From these results it is seen that the degree of precipitation of the colloidal paraffin does not vary directly with the amount of protein in the cerebro-spinal fluid, but that the relationship is more complex.

Nonne-Apelt and Pandy tests.—Every case giving a positive Pandy or Nonne-Apelt result, with the few exceptions quoted below, gave abnormal curves with the colloidal paraffin reaction.

Disease	Nonne-Apelt	Pandy	Colloidal Paraffin B
Cerebral tumour	..	Very weakly +	0001000000
Cerebral tumour	+	+	0001000000
Cerebellar tumour	+	+	A.0000010000 B.0000000000
Uræmia	Very weakly +	Very weakly +	0010000000
Progressive muscular atrophy..	Negative	Very weakly +	0000000000
Bulbar palsy	Negative	Very weakly +	0000000000

Wassermann.—No case with a positive Wassermann reaction in the cerebro-spinal fluid gave a normal reading with the colloidal paraffin. One case of bulbar palsy with the Wassermann reaction in the cerebro-spinal fluid negative, and in the blood 4320, gave an absolutely negative

colloidal paraffin result. A fluid taken from a patient diagnosed as a case of tabes gave a Wassermann reaction in the blood 2100, and in the cerebro-spinal fluid 1000; in this case the Lange was negative and the colloidal paraffin A gave a slight precipitation in the fourth and fifth tubes.

A positive Wassermann is not necessary to ensure a paretic or luetic curve, as the cerebro-spinal fluids in disseminated sclerosis demonstrate.

Lange test.—A striking similarity is at once apparent on comparing the results of the Lange and colloidal paraffin tests; especially is this noticeable in the cases of disseminated sclerosis. Disseminated sclerosis may be considered a good test for the sensitiveness of a colloidal reagent. Carmichael and Greenfield have found that the Lange reaction gives a paretic curve in 10 per cent. and a tabetic curve in 19 per cent. of cases of disseminated sclerosis. It is to be noticed that with the benzoin tests investigators have found the curves less marked, showing that benzoin is not so sensitive as gold. The colloidal paraffin reaction has not been performed on a large enough number of cases of disseminated sclerosis to justify the taking of percentages, but the cases which have been examined by this reaction follow closely the results obtained with that of Lange, demonstrating that the two reactions are equally sensitive.

By comparing the relative merits of the colloidal gold and colloidal paraffin tests, it is seen that the paraffin reaction has many points in its favour:—

(*a*) It is cheaper.

(*b*) The technique of the paraffin test is simpler.

(*c*) The solution does not deteriorate so easily as colloidal gold.

(*d*) The actual reading of results of the paraffin tests is easier and more reliable than the colour test of the Lange reaction, which allows for considerable diversity of opinion.

(*e*) It has been found that different batches of gold solution are more sensitive than others, so that the same cerebro-spinal fluid may cause precipitation as high as "3" with one batch and only up to "2" or "1" with other batches. This varied sensitiveness has not been encountered in any of the paraffin solutions.

Colloidal benzoin and colloidal gamboge tests.—I was not able to do an examination of the cerebro-spinal fluid by these methods until the last few fluids, when I availed myself of the opportunity to make an examination of the cerebro-spinal fluid by these reactions for the purpose of comparison. The comparative results of this examination are as follows:—

Disease	Colloidal benzoin	Colloidal gamboge	Colloidal paraffin A	Colloidal paraffin B	Colloidal paraffin C
Disseminated sclerosis	..	10000	0110000000	0000001000	0111000000
Neurosis	00000	..	0000000000	0000000000	0000000000
G.P.I.	12222	22210	2444431100	4442100000	3442000000
G.P.I.	12222	22210	1444310000	2432100000	3431000000

The results show that the colloidal paraffin reaction gives very similar readings to the colloidal benzoin and gamboge tests. The paraffin reaction only requires 0·25 c.c. cerebro-spinal fluid, while the colloidal benzoin test needs 1·75 c.c. cerebro-spinal fluid, which is a point in favour of the former test.

As an aid to diagnosis the colloidal paraffin reaction is valuable and could easily be made a test in the routine examination of cerebro-spinal fluid, as it requires so small a sample. The technique is simple and no great skill is required in its performance. As regards the various modifications of the colloidal paraffin test, I am now in a position to give an opinion as to their relative advantages.

Kafka's original method.—The chief objection to this method is that it is too sensitive, a small amount of wax comes down even in the control, and the readings are rendered difficult.

Colloidal paraffin B.—This modification is very satisfactory, no wax is precipitated in the control, but the dilutions differ from those used in the routine Lange test and comparisons cannot be made on the same sample.

Colloidal paraffin C.—This method is perhaps the most satisfactory of those employed in this series of experiments. It has the advantages of not being hypersensitive, and also of containing cerebro-spinal fluid in dilutions suitable for comparison with the Lange test.

The most efficient technique for the reaction seems to be modification C., and on the whole the most efficient working paraffin solution one containing one part of stock solution to two of water.

In conclusion, I should like to express my appreciation and gratitude to Dr. Geoffrey Hadfield of the Bristol General Hospital, and to Dr. Greenfield and the physicians of the National Hospital, Queen Square, London, for their great kindness in supplying material and apparatus for my investigations.

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Proceedings of the Section of Neurology of the Royal Society of Medicine.

President—Dr. H. CAMPBELL THOMSON.

Clinical Meeting held at the National Hospital for the Paralysed and Epileptic, Queen Square, W.C.1, on Thursday, March 11, at 8 p.m.

A Case of Chronic Progressive Polyneuritis with Unusual Distribution of Sensory Loss.

By Dr. C. P. SYMONDS.

D. H., aged 22, states that she had diphtheria at the age of 6, and has ever since suffered from weakness of the lower limbs. This was not so severe as to prevent her from attending school, but she was unable to take part in any form of sport on account of the unsteadiness of her legs. On the whole she thinks the disability was slightly progressive. She did not notice any disability in the upper limbs until after she took up the occupation of waitress two years ago. Weakness and unsteadiness of the arms subsequently became so marked as to lead to constant breakages and the loss of her situation. During the past year or so she has suffered a good deal from cramps in the calves, and has had peculiar cutaneous sensations in both antecubital fossæ, which she describes as the skin "feeling sticky." On examination there is no defect of cerebral function, special senses or cranial nerves. There is generalized weakness, without gross wasting, of all four limbs, with absence of tendon-jerks, save for the knee-jerks, which are both sluggish and less easily obtained on the right. The abdominal reflexes are present, the plantar responses absent. There is relative anæsthesia to cotton wool and pin-prick from elbows to wrists, and knees to ankles, the hands and feet escaping. Sense of position is poor in the toes, fair at larger joints. Vibration sense absent over the tibiæ. No loss of sphincter control. Gait, high stepping on a wide base. Wassermann reaction negative in blood and spinal fluid. Cells and protein and colloidal benzoin curve normal in the latter. Test meal shows an acid-resisting juice and slight hyperchlorhydria. Urine contains no sugar. No evidence obtained of any chemical poisoning.

A Case of Lead Palsy with Spastic Paraplegia.

By Dr. C. P. SYMONDS.

C. S., aged 53, first presented himself at hospital two years ago for weakness of the limbs. He had worked as a solderer for thirty years, being constantly

exposed to the fumes of lead, but without ill effect, save for constipation and occasional colic. For several months previous to the present illness he had become over-conscientious at his work, and had spent his evenings and week-ends in the workroom.

Weakness was first noticed in the right hand two months before he was seen at hospital. He then experienced "cramps" and weakness in the right leg; the left limbs were subsequently affected in a similar fashion.

On examination he showed a typical blue line on the gums, and the blood showed a moderately severe anæmia with punctate basophilia.

In the nervous system no abnormality of special senses or cranial nerves was observed. The upper limbs presented the classical picture of lead palsy; atrophic paralysis of the long extensors of wrists and fingers, with some weakness and wasting of the shoulder-girdles and intrinsic muscles of the hands. The supinator longus on both sides was spared. The lower limbs were weak, especially in movements of flexion, but not wasted. Both were slightly spastic in extension, with increased tendon reflexes and sustained ankle clonus. The plantar response on the right was extensor, on the left indefinite. The abdominal reflexes were absent, save for an occasional twitch in the right lower quadrant. The supinator jerks were not obtained. There was no sensory loss nor any complaint of paræsthesiæ. Sphincter control was intact. The gait was spastic.

The Wassermann reaction was negative in blood and spinal fluid. The latter showed 0·07 per cent. protein and 12 lymphocytes per c.mm.

Under treatment this patient has made an almost complete recovery so far as the upper limbs are concerned. The lower limbs, however, are still somewhat weak and spastic, with increased tendon reflexes and ankle clonus, and the difficulty in walking remains a serious disability.

Dr. FEILING mentioned a case of amyotrophic lateral sclerosis in a lead worker, with chronic interstitial nephritis.

Dr. HARRIS said there was no doubt that both subacute and chronic poliomyelitis occurred as well as polyneuritis. The subacute and chronic forms showed no tendency to recover.

Dr. CLOAKE asked for evidence in the first case of chronic diphtheria or chronic sepsis.

Dr. GREENFIELD mentioned a patient with three recurrent attacks of polyneuritis, each time associated with the presence of Klebs-Löffler bacilli in the nasopharynx.

Dr. HARRIS asked for any evidence of myasthenia in the first case.

Dr. C. P. SYMONDS said there were no myasthenic symptoms, but the nose and throat had not been examined bacteriologically. The patient with lead palsy had, when first under observation, wasting of hand and shoulder muscles and has since recovered. The author was sceptical as to whether any of these lead cases were polyneuritic in the strict sense, and had yet to hear of loss of sensation or paræsthesia.

Case of Myotonia Congenita.

By Dr. REDVERS N. IRONSIDE (for Dr. F. M. R. WALSHE).

J. G., aged 22. No family history of nervous disease or disease similar to this obtained. Patient first noticed eighteen months ago that at times his legs become stiff and rigid when he stands or walks and the rigidity passes off in a few minutes. This occurs several times a day. He has fallen during these spasms. Suffered from poisoned hand in April, 1925.

On examination.—Discs, pupils and ocular movements normal. Generalized and marked enlargement of trunk- and limb-muscles. Herculean torso and limbs. Marked weakness of right arm and hand, slight of left arm. Marked myotonia of all limbs and trunk muscles. Myotonia of the tongue and in the facial muscles. Doubtful wasting of clavicular portion of sterno-mastoids. Tendon reflexes brisk, no delay in relaxation. Plantar responses flexor. No sphincter disturbance.

Dr. IRONSIDE pointed out that this case recalled the condition of "hypertrophia musculorum vera." Spiller, in *Brain* of 1913, quotes Wood's case, in which painful muscular cramps occurred. The atrophy of the sterno-mastoids seemed to be unique, and suggested that there might be transitions between myotonia with hypertrophy of muscles and dystrophia myotonica.

Referring to the pathogenesis of myotonia, the speaker pointed out the resemblance of the muscular spasms to those which occurred in veratrin poisoning and on stimulation of the sciatic nerve in animals after injection of sodium phosphate. He recalled the "intention spasms" of tetania parathyreopriva, and speculated as to whether there was any connection between tetany and myotonia.

Dr. ADIE said this case reminded one of three distinct muscle diseases (dystrophia myotonica, hypertrophia musculorum vera and Thomsen's disease). He suggested that this case was one of hypertrophia musculorum vera with muscular atrophy, a disease which should be included under the heading of myopathy.

Dr. BARNES referred to a family of eighty-four members, one of whom suffered from hypertrophia musculorum vera. Thirty-six of them showed unusual types of myopathy (including two "distal" cases). One case had no perceptible sterno-cleido-mastoids. One had myotonic manifestations.

Case of Spondylitis of Upper Cervical Vertebræ, including Atlas and Axis, with Cord Compression. ? Typhoid Origin.

By Dr. REDVERS N. IRONSIDE (for Dr. F. M. R. WALSHE).

T. D., aged 54. Patient in 1890 had an illness called "enteric" at Malta. Had typhoid fever in 1908, and an illness diagnosed as typhoid in 1910 in Northern Ontario. Influenza with pneumonia and left empyema in 1920 (operation). Slight winter cough for six years. Seven years ago he began to drop things from his left hand and noticed occasional jerkings of his head.

Six years ago began to notice a hard lump on the back of his neck on the right side, and his neck felt stiff. Had to wear a larger collar. Three years ago weakness of the right upper limb. One year ago unsteadiness in walking, frequency of micturition, pins and needles in right elbow, and his voice becoming fainter. Occasional pain in inguinal regions. No abscesses, no pyrexia.

On examination.—Large reddish swelling in the cervical and suboccipital regions on the right side. Neck movements restricted. Much coarse crepitus and grating when head is moved. Pupils small, right slightly the larger; they do not dilate to shade but react to light. Nystagmoid movements to right and left. Jaw deviates to left. Weakness and wasting of extensors of both elbows and supinators of both wrists. Grips good. Thoracic wall immobile. Abdominal wall spastic. Legs spastic in extension. Wasting of muscles of lower limbs.

To cutaneous warmth and pressure pain, incomplete sensory loss over C. 1 to 5 on the right side, and C. 1 to 4 on the left. Hot and cold are not differentiated below C. 2 level, but the responses are better on the left half of the trunk and limbs than over the right. Vibration sense absent over radial styloids and tibial malleoli. Sense of passive movement impaired in upper and lower limbs.

Reflexes: Jaw-jerk absent. Tendon reflexes very brisk, except right triceps-jerk, which is feeble. Abdominals present. Plantar responses both extensor.

Frequency of micturition and difficulty in commencing micturition. Cerebro-spinal fluid clear. Protein 0·15 per cent. Nonne-Apelt positive. Widal positive 1 in 6 in cerebro-spinal fluid; positive 1 in 1,000 in blood.

Wassermann reaction negative in blood and cerebro-spinal fluid. No other than coliform colonies grown from the stools.

X-ray shows gross bony deformity (kypho-scoliosis) of upper cervical vertebræ.

Dr. IRONSIDE stated that the interest lay in the high cervical site, and the history of three attacks of typhoid fever. He suggested that *Bacillus typhosus* might lie dormant in the bone marrow for years, just as it is known to do in the gall-bladder. The literature shows that typhoid spine may appear some time after the acute attack and cause great deformity, and even cord compression. The bony lesion may be an osteomyelitis. The mobility of the spine, the lack of abscess formation and the recovery rate were all against an adult Pott's disease.

Dr. GREENFIELD referred to a case of myelitis due to osteomyelitis of the vertebræ. The cerebro-spinal fluid in Dr. Ironside's case was of interest in demonstrating: (1) a positive Widal reaction; (2) presence of complement; and (3) high protein content.

Dr. ADIE referred to a case of so-called gumma of the skull which turned out to be a typhoid abscess occurring some years after enteric fever. He recommended that a specimen of bile be obtained from Dr. Ironside's case by duodenal drainage, and searched for the presence of typhoid bacilli.

Two Cases of Disseminated Sclerosis with Vasomotor Changes.

By Dr. REDVERS N. IRONSIDE.

Case A.—A. C., aged 45. Onset of symptoms seven years ago (aged 38), with readily tiring, dragging and numbness of the left lower limb. Transient diplopia has since recurred at intervals. Frequency of micturition. Three years ago clumsiness and weakness of the left upper limb.

Previous history.—"Veins taken out" of right leg for varicose ulcer four years ago. "Patch always"—cough, fever, and loss of weight for several months in 1916. Four children—youngest has tuberculosis. Mother, aged 72, in hospital now because of weakness of arms and legs of several years' duration.

On examination.—Left paracentral scotoma in nasal half of field. Optic discs pale, left more than right. Fine nystagmus on lateral deviation of eyes to left and right. Left lower facial asymmetry. Weakness of both upper limbs, greater in the left than right; left upper limb hypertonic, "lead pipe variety." Action tremor in left leg more than right. Both legs weak and spastic, left weaker than right. Sensory loss to cutaneous warmth and pressure pain over legs, trunk and arms to C. 4 level. Vibration absent on malleoli, present in arms. No loss of postural sense. Tendon reflexes very brisk, abdominal reflexes absent. Plantars, right extensor, left extensor. Frequency, urgency and difficulty in micturition. Slight right dorsal scoliosis. Cerebro-spinal fluid normal. Wassermann reaction negative in blood aud cerebro-spinal fluid.

For the past two years patient has noticed at times a deep purple discoloration of the left foot and lower half of the left leg. Slight swelling. At times the foot is cherry red, at times almost black. It is quite painless.

Case B.—G. S., aged 39. Three years ago she began to drag her left foot. Tires easily. During the past year urgency and frequency of micturition. No diplopia. Periods now regular, but ten years ago had amenorrhœa lasting ten months. During this time she used to lose consciousness for a few seconds at intervals of three to four months, following a flushing of the face.

On examination.—Visual acuteness $\frac{6}{12}$ right and left. Marked temporal pallor of the right optic disc. Left optic disc pale (medullated nerve-fibres also). Nystagmoid jerkings of eyes on extreme right lateral deviation. Slight action tremor right and left. Legs very spastic, left more than right. Tendon reflexes brisk. Plantar responses extensor. Abdominal reflexes elicited, but left tires easily. Vibration and postural sensibility impaired in lower limbs, normal in upper limbs. Cerebro-spinal fluid normal. Wassermann reaction negative in blood and cerebro-spinal fluid. Hands succulent. Fingers either bright scarlet or bluish black. Subungual hæmorrhages in the third left finger. Nails transversely fissured. Frequent chilblains.

Dr. IRONSIDE stated that these two patients recalled the ten cases of erythromelalgia occurring in spinal cord disease reported by Collier in 1898. At least five of these cases suffered with disseminated sclerosis, and in one of the tabetic cases the erythromelalgia was painless.

With regard to the site of the lesion, Lannois and Porot have described in a case of erythromelalgia degeneration of the intermedio-lateral cells, the spinal origin of the preganglionic sympathetic fibres.

Dr. FEILING queried the term "erythromelalgia" for a painless disorder; the coldness of the limb he stated was unlike the heat of erythromelalgia.

Dr. IRONSIDE suggested that the loss of cutaneous sensibility might account for the painlessness.

Case of Quadriplegia; ? Progressive Muscular Atrophy.

By Dr. REDVERS N. IRONSIDE (for Dr. T. GRAINGER STEWART).

E. C., aged 41. Four years ago had "influenza" during pregnancy, followed by cramps in thighs and difficulty in walking. No sore throat. Eighteen months later, left foot-drop. Six months later right foot-drop. Noticed quivering of muscles of thighs at this time. One year later became bedridden, noticed she could not lift her arms at the shoulders, although hands were strong. Lately weakness of back muscles; patient unable to sit up unless propped up.

Rheumatic fever aged 9. Has never had diphtheria.

On examination: Pupils react. Complete flaccid palsy of both lower limbs with only slight muscular wasting. No fibrillation. Weakness of trapezii and deltoids on the two sides, and of the pronators of the right wrist, right triceps, and left biceps and triceps, supinators of left wrist and flexors of fingers. Left latissimus dorsi does not contract on coughing. Upward deviation of umbilicus of 1 in. Slight wasting of deltoids, supra- and infra-spinati, trapezii and erector spinæ glutei and vastus internus on the two sides. No fibrillation seen. No sensory defect. No tenderness of muscles. All tendon reflexes absent. Plantars absent at toes. No sphincter defect.

Muscles of lower limbs show complete reaction to degeneration. In the upper limbs either normal or partial reaction to degeneration.

Cerebro-spinal fluid normal. Wassermann reaction negative in blood and cerebro-spinal fluid.

Case of Encephalitis Lethargica followed by Myelitis.

By Dr. C. M. HINDS HOWELL.

A. A., male, aged 40. Admitted January 30, 1926, with a two weeks' history of pain in the back, difficulty in walking of one week's duration and precipitant micturition for three days. Two days before admission he had cramp-like spasmodic pains in his right leg and occasionally in the left.

Family history.—Wife had three miscarriages. No living children.

Past history.—In November, 1925, patient became lethargic, fell asleep over his meals; his eyes continually blinked. He felt giddy and vomited on several occasions. By December his condition had improved.

In January, 1926, the pain in the back commenced in the upper dorsal

spine and radiated into the right shoulder. Pain of girdle character was also complained of below the costal margin.

Cranial nerves.—Nothing abnormal.

There was some rigidity of neck. Unable to sit up on account of pain in back. Left arm weaker than right and showing slight generalized wasting. Reflexes: right brisker than left.

Both legs hypertonic, left more than right. Loss of power in both, more marked in left. Deep reflexes exaggerated. Right plantar response equivocal, left extensor. Clonus at both ankle-joints. Abdominal reflexes absent.

Sensation.—Impairment to cutaneous warmth and pressure pain in whole of right leg, thigh and trunk, and some impairment to pressure pain in right arm. There was a zone of hyperæsthesia extending round the body at level of D. 8 and 9. Other forms of sensation normal.

Retention of urine.

Three weeks later the sensory changes over thorax and abdomen could not be detected, but the impairment to pressure pain and cutaneous warmth remained in leg and thigh and arm. Power in legs and arm had improved.

Cerebro-spinal fluid clear and colourless. 10 cells per c.mm., about 10 per cent. large mononuclears. Total protein 0·04 per cent. Nonne-Apelt very weakly positive. Pandy: weakly +. Lange: no change in any tube. Wassermann reaction negative in blood and cerebro-spinal fluid.

Case of Hereditary Ataxia.

By Dr. ARTHUR J. DAVIES (for Dr. RISIEN RUSSELL).

R. J., male, aged 14. Four years' history of tremor in right hand when attempting to use it, coincident with a right-sided squint. Nine months ago, awkwardness of the right foot. Patient's symptoms have been steadily progressive; said to be duller mentally of late.

Family history.—Six other children alive; the eldest son is said to "walk a little peculiarly." The eldest daughter has a congenital coloboma of the iris. One other brother is said to have an awkward gait. Three still-births. Mother and father healthy.

Previous history.—Head was remarked to have been big at birth. Nine years ago had pneumonia; he thinks "he has never been quite well since." Fundi normal: strabismus, right eye; nystagmus, on lateral ocular deviation. Other cranial nerves normal. Intention tremor and dysdiadokokinesia in right arm. Right leg is hypertonic with slight general wasting; both feet are in attitude of pes cavus, especially the right. No ataxia. No sensory disturbance. Knee- and ankle-jerks brisk, right more than left. Ill-sustained right ankle clonus. Both plantar responses flexor in type. Blood Wassermann reaction negative.

Dr. A. J. Davies suggested that the case was one of the well recognized clinical group of cases intermediate in type between a Friedreich's and a Marie's ataxia.

Case of Myopathy with Fibrillation.

By Dr. ARTHUR J. DAVIES (for Dr. RISIEN RUSSELL).

D. M., male, aged 30. Fifteen years' history of progressive weakness and wasting of muscles. At the age of 6 he was noticed to tire very easily. Legs were affected first. About four years ago right arm was noticed to be wasted and weak, the left about two years ago.

Family history.—Sister suffering from similar condition. A sister died aged 4, and never walked; two others healthy. Mother and father healthy. Nothing abnormal in the cranial nerves. Wasting of muscles of shoulder-girdle, arms and forearms, more marked on right. Ribs prominent. Proximal muscles of legs wasted. The tibial and calf muscles show hypertrophy. Fibrillation has been seen in muscles of the shoulder-girdle. All deep reflexes absent, both plantar responses flexor in type. Abdominals present. No sensory disturbance. Gait typically waddling. "Climbs up himself" when rising from the floor. Some precipitancy of micturition.

Dr. A. J. DAVIES referred to a similar case described by Dr. J. Taylor in 1903, in a boy, aged 11, suffering from pseudo-hypertrophic muscular dystrophy.

Dr. BARNES thought the fibrillation was coarser than the usual variety, more like myokymia. He has only seen one other case.

Dr. SYMONDS said he had shown a case of pseudo-hypertrophic muscular dystrophy with coarse fibrillation in the hypertrophied calves.

Dr. ADIE referred to occasional fibrillation in dystrophia myotonica.

Case of Myasthenia Gravis.

By Dr. ARTHUR J. DAVIES (for Dr. RISIEN RUSSELL).

A. B., female, aged 50. Twelve months' history of drooping of eyelids, difficulty in speaking and chewing foods; on one or two occasions regurgitation through nose. No diplopia. Condition varies, always worse towards night and better some days than others.

Family history.—Nil ad rem.

Previous history.—Nil ad rem.

Speech nasal in character; difficulty in pronouncing labials and dentals. Fundi normal. Ocular movements normal. Weakness of levatores palpebrarum, particularly left. Weakness of masseters and temporals. Lower jaw cannot be closed. All facial muscles weak, particularly on left side, the orbicularis oris especially is affected. Palatal movement impaired, and uvula pulled over to right. Lateral movements of the tongue weak; no wasting. Head tends to fall about on shoulders. Slight scoliosis. Sits up in bed by rolling over on to left side and pushing herself up. No sensory disturbance. Nothing abnormal in arms and legs. Tendon reflexes brisk. Abdominals absent, both plantar responses flexor in type.

Dr. A. J. DAVIES said the diagnosis lay between myasthenia gravis and pro-

gressive muscular atrophy. Against the latter was the onset with ptosis. Since coming under observation the patient has shown very marked daily variation, being much worse towards nightfall. There is no difficulty in deglutition. Cerebro-spinal fluid was normal, except that the glucose was 0·104 per cent.

Dr. GREENFIELD referred to two cases of myasthenia examined lately with sugar about 0·10 per cent.

Dr. SYMONDS said that sugar tolerance is lowered in myasthenia gravis.

Dr. CLOAKE pointed out that the sugar content in the cerebro-spinal fluid is raised one hour after meals.

Case of Post-encephalitic Respiratory Disorder.

By Dr. MACDONALD CRITCHLEY (for Dr. C. M. HINDS HOWELL).

E. B., male, aged 31, had an attack of acute epidemic encephalitis in 1924, and states he has never felt well since. Six months ago he began to feel sensations of "rawness" behind his knees, associated with quickness of breathing.

Physical examination revealed a state of mild Parkinsonism. The respiratory disorder consists in paroxysms of noisy tachypnœa in which the respiration rate becomes as great as fifty per minute. He stands—during the attack—in a characteristic bowed attitude, swaying backwards and forwards with each respiration. After about thirty to fifty seconds he draws himself up and leans far back; the tachypnœa is now succeeded by a spell of full inspiratory apnœa lasting about fifteen to twenty seconds. The face and neck become suffused, but there is no clouding or loss of consciousness. The apnœic phase is terminated by a prolonged expiration. These paroxysms come on at any time, but are more frequent towards nightfall. They tend to be more marked when under emotional stress. Vigorous external stimuli are often capable of arresting or diverting an attack. There is no inversion of the sleep rhythm and no conduct change, but there are possibly occasional hallucinations of hearing.

Cerebro-cerebellar Diplegia with Mirror Writing.

By Dr. MACDONALD CRITCHLEY (for Dr. RISIEN RUSSELL).

Girl, aged 6. Was backward in sitting up and learning to walk. Her gait has always been very unsteady and there is some tremulousness of the arms. No mental impairment. Right handed.

Family history and previous health.—Nil ad rem.

Head somewhat enlarged; nystagmus on lateral deviation of the eyes. Slight intention tremor of both arms. Gait shows a cerebellar type of ataxy. All tendon-jerks are brisk and there is a bilateral extensor plantar response. The child writes "mirror fashion" with her left hand.

Dr. Critchley showed cinematograph films of these two cases.

Dr. ADIE asked whether there was any evidence of congenital word blindness in the patient who wrote mirror fashion.

Dr. CRITCHLEY replied that he believed that some degree of congenital word blindness existed in this patient. The child could read a few simple words, such as "cat" or "dog," and she could read them equally well if written mirror-wise or in a dextrad fashion. The speaker believed that most diplegics who were of sufficient intelligence to write, wrote mirror fashion with the left hand. The explanation of this was still a matter of hypothesis. He suggested that a normal infant learns to write primarily by means of its kinesthetic sense. Thus it learns to associate a particular muscle movement with a particular letter or word. The innervation of identical neurons in the same sequence, upon the opposite hemisphere, produces a mirrored reproduction of the original. At a late stage in its education, however, the child learns, by training the visual perception, to associate particular symbols with such and such a word; it also learns that the correct direction for reading and writing is from left to right. In this way a visual memory develops. In diplegics, amongst others, this visual sense develops late if at all; consequently, when a pencil is placed in the left hand the child writes with its kinesthetic memory sense, and hence produces mirror writing. Mirror writing, he suggested, is the normal mode of writing with the left hand before the education of the perception.

Dr. SYMONDS asked whether any periodicity was observed in the onset of the respiratory disorder. He referred to a patient of his own who showed nocturnal aggravation of tachypnœa.

Dr. CRITCHLEY stated that it is well known that many of these cases of respiratory disorder exhibit a diurnal periodicity in appearance. The present case also showed this periodicity, for the attacks of tachypnœa became much more frequent and much more severe towards nightfall.

Progressive Muscular Atrophy due to Lead.

By Dr. MACDONALD CRITCHLEY (for Dr. S. A. KINNIER WILSON).

Male, aged 48, has been a coach painter for over ten years, using in his employment large quantities of lead paint. His present disability consists in weakness, cramps and fibrillation in the muscles of the legs. Symptoms commenced three years ago and are getting progressively worse.

There is definite weakness with atrophy of the muscles of the right leg; fibrillation is visible in muscles of the upper and lower extremities. Abdominal reflexes present. Knee and ankle jerks obtainable; plantar responses are equivocal on the right side and extensor on the left. No sensory changes. Blood-pressure 100. Blue line on gums. Anæmia present with polychromasia and basophil stippling of erythrocytes. No abdominal symptoms. No albuminuria.

Muscular Atrophy following Diphtheria.

By Dr. MACDONALD CRITCHLEY (for Dr. S. A. KINNIER WILSON).

J. P., male, aged 17, had an attack of diphtheria seven years ago, followed four months later by weakness in the right leg. The weakness progressed and extended to his other leg and later to his shoulder-muscles and trunk. His condition has been steadily progressive over the last seven years.

Family history.—Three sisters alive and well. Mother healthy; father drowned.

There is gross wasting and weakness of the muscles of the shoulder girdle, of the thighs and, to a lesser extent, of the trunk. No fibrillation visible. Tendon-jerks absent. No sensory loss. Scoliosis present. Definite "wasp-waist" deformity. Some winging of scapulæ; patient tends to slip through one's hands when one attempts to raise him by his armpits. Cranial nerves normal except for some difficulty in whistling. No sphincter disturbance.

Dr. CRITCHLEY suggested that his patient was a myopathy of the Buzzard type. Other cases of muscular dystrophy arising after diphtheria are on record, but their rarity suggested that they were merely coincidental.

Notices of Recent Publications.

A Classification of the Tumours of the Glioma Group on a Histogenetic Basis with a Correlated Study of Prognosis. By Percival Bailey and Harvey Cushing. Philadelphia, London and Montreal: J. B. Lippincott Company. Pp. 175.

This monograph is the outcome of a special investigation on 400 gliomata collected during a period of twenty-one years at the Johns Hopkins and the Peter Bent Brigham Hospitals. These tumours were studied by special neuroglial staining methods, and also, when the appearances seemed to call for it, by neurofibril stains. By these means and by a study of the embryology of neural and neuroglial tissue, the gliomata and allied tumours have been separated into sixteen classes, according to the similarity of the predominant tumour cell to some stage in the differentiation of the cells arising from the neural groove.

As a preliminary grouping it was considered that tumours might arise either from cells which had not yet been differentiated into neuroblast and spongioblast, or from those which had been so differentiated. The latter again naturally divided themselves into nerve-cell tumours and tumours arising from neuroglial, ependymal or pineal cells. The tumours in each of these groups might reproduce either the first stages of differentiation or the more fully differentiated cells. Thus the authors separate the neuroblastoma from the ganglioneuroma, the pineoblastoma from the pinealoma, and the spongioblastoma from the astrocytoma. This classification is chiefly theoretical, but it was found that some tumours of the series fitted more or less exactly into each class. It is not claimed that each tumour will correspond exactly to one stage in the differentiation of the central nervous system. This would be unlikely on theoretical grounds, and practically it was found that in some parts of a tumour the cells were more differentiated than in another part, and also that when a tumour continued to grow after operation it might revert to a more rudimentary cell type.

The last part of the book consists in an attempt to estimate, by reference to case histories, which types of tumour were more malignant, and which were relatively benign. In some of the classes it was impossible to tell this with certainty as only one or two specimens of that type were available, but as a general rule it was found that the more exactly a tumour tended to reproduce a fully differentiated cell type the slower did it grow and the less tendency did it have to recur after operative removal.

The book is of great value as a record of original work on an important

subject, and marks a distinct advance in the histology of tumours of the central nervous system. We regret that the description of some of the different classes is not fuller, and that for some the reader is referred to earlier publications in medical journals. Sometimes it is not altogether easy to form a clear conception of the criteria on which the differentiation is based; for example, the astroblastomata and protoplasmic astrocytomata appear to resemble one another very closely both in the description and in the illustrations given. An extensive bibliography redeems these shortcomings and increases the value of the book as a groundwork for further research.

The Clinical Examination of the Nervous System. By G. H. Monrad-Krohn. Third Edition. London: H. K. Lewis. Pp. 201. Price 7s. 6d.

The successive editions of this well-known book have become more and more complete. This, the third English edition, has been thoroughly revised and a certain number of additions have been made to the original text. New illustrations are also provided. The clear and simple form of the text, and the careful but adequate description of the modes of testing the functions of the nervous system in health and disease, make the book eminently suitable for the student; and even neurologists in isolated places can from it learn the most recent advances in the methods of investigation of nervous diseases.

Brain and Spinal Cord. By Emil Villiger. English Translation by W. H. F. Addison. Third American Edition. Pp. 335, with 262 illustrations. London: Lippincott Company. 1926. Price 25s.

This new edition of the American translation of Villiger's book on the anatomy of the nervous system has been considerably enlarged; a certain amount of the old text has been rewritten and brought up to date, and new sections have been added on the sympathetic system and on the neuroglia. Both of these sections, and especially that on the neuroglia, might be with advantage further amplified. The number of illustrations is also increased.

The character of the book is well known; it has been undoubtedly of service to students, and the emphasis laid in its later editions on the clinical significance of many anatomical facts has certainly helped beginners in neurology. That this has been the main object of the author is evident from the numerous diagrams it contains; many of these are unnecessarily crude and must fail to give an adequate idea of the structural relations of the parts indicated in them.

The feature of the book that is most open to criticism is the appropriation of space to separate portions of the nervous system; several pages are, for instance, devoted to the rhinencephalon, while the structure and connections of the corpus striatum, which is assuming a great importance in clinical neurology, are given only a few lines of description.

Die Bedeutung der Roten Kerne und des übrigen Mittelhirns für Muskeltonus, Körperstellung und Labyrinthreflexe. Von G. G. J. RADEMAKER. German Translation by E. LE BLANC. S. 340. Berlin: J. Springer. 1926. Price 27 Mark.

This book, which was originally published in Dutch, has been already reviewed in this Journal (1924, 47, 390). The German translation, which will make it more easily available to English readers, has been well carried out, in fact the only complaint that can be brought against it is the absence of an index, by which reference to any question under investigation would have been made simpler.

A few additions have been made, especially to the chapters on the physiology and pathology of the midbrain, and the number of illustrations has been increased. It is an important, almost necessary, supplement from the anatomical side to much of the work published by Magnus and his school.

Les Syndromes des Nerfs de la Queue de Cheval. Par J. N. PÉRON. Pp. 209. Paris: A. Legrand.

This small monograph from Professor Guillain's clinic at the Salpêtrière is devoted to diseases and injuries of the cauda equina, excluding wounds and congenital malformations. There is a short chapter on the anatomy of the lowest segments of the cord and on the relations of the nerve-roots connected with them.

The clinical symptoms are then considered in separate chapters on tumours of the cauda equina and of the soft parts around it, lesions of the bones involving the roots, and inflammatory lesions, including syphilitic diseases. Nothing new of importance is brought out in its pages, but the clinical and pathological aspects of a large number of cases are recorded, and there are some interesting illustrations.

Le Syndrome chiasmatique: étude clinique pathogénique thérapeutique. Par A. FAVORY. Pp. 184, with 66 illustrations. Paris: O. Doin. 1926. Price 25 fr.

One of the most interesting features of this book is the careful study of the anatomical relations of the optic chiasma, especially its relations to the pituitary body and the larger arteries of the base of the brain. This, it is true, is not entirely new, as De Schweinitz and others have previously described them, but it is valuable to have the earlier observations confirmed and illustrated so plainly as they are by the author. The importance of thorough examination of the fields of vision is emphasized, and especially the use of graded stimuli. In the chapter on the treatment of chiasmal lesions the good results obtained by the application of X-rays to pituitary tumours is brought out. The symptoms produced by different types of lesions are illustrated by the case histories of a large number of patients who came under the author's observation.

Les Grands Narcotiques Sociaux. Par Dr. LEGRAIN. Pp. 459 Paris: Maloine. 1925. Price 20 fr.

The three social narcotics, or "poisons of the intelligence," dealt with in this volume are opium, alcohol, and tobacco. On opening the book, which is bulky and pretentious, one had hoped to find a clear analysis of the actions of these drugs from the physiological and psychological points of view, as well as from their social aspects, but instead it consists mostly of page after page of rhetorical denunciation of their use, such as we are accustomed to meet in the propaganda of all cranks. Tobacco, for instance, is merely described as a stupefying narcotic which affects profoundly the integrity of the cerebral cells, while the author states that smoking usually gives no pleasurable sensation, and is only a manifestation of a habit epidemic. The use of tobacco leads to loss of the æsthetic sense and of propriety, while the smoker is characterized by an enormous enfeeblement of will. The book can claim no medical interest.

Histological Technique for Normal Tissues, Morbid Changes and the Identification of Parasites. By H. M. CARLETON. (Chapters VII and VIII in collaboration with FREDERICK HAYNES.) Pp. 398. Humphrey Milford: Oxford University Press. 1926.

This book does not attempt to give a description of every known method of fixing, cutting or staining normal and pathological tissues. Rather it is intended to form a groundwork for sound histological study. As an introduction to this the first chapter, which deals with the intimate structure of the cell, is a useful innovation. We are also glad to see that a clear distinction is made between micro-anatomy and cytology, a distinction which the neuro-histologist has long recognized, but which is often forgotten in the examination of bodily organs other than the nervous system. The author makes it clear that in studying micro-anatomy, that is the topographical relation of one tissue to another, methods of fixation and staining must be employed which are not always those best suited to the finer study of the cells.

The chapters on fixation and on cutting paraffin sections are excellent. The descriptions of celloidin and frozen section work are not so good. It appears, however, that the author has only used the cruder forms of celloidin microtome, for he says that it is never possible to cut sections in celloidin at less than 10 μ, "while 15 to 20 μ is the usual thickness." The celloidin microtome illustrated is a large Schantze model, for which the above sentence is no doubt true.

Another fault in this part of the book is that the word "formol" is definitely used in two different senses. Thus on p. 31 we are told that formol is 40 per cent. formaldehyde gas, and in a footnote that "20 per cent. formol equal parts of commercial formalin and water."

In the section on common staining methods we are surpr[illegible]ed to find no description of Weigert's iron hæmatoxylin or of alum carmine, both of which

are surely among the most commonly used formulæ for these stains. Nor is it made clear that in staining with van Gieson's solutions the water, dehydrants and mordanting media must be neutral or acid if the stain is to be preserved.

Of the special staining methods those for the nervous system concern us chiefly. This section is disappointing, as none of Alzheimer's neuroglia methods are included. Busch's modifications of the Marchi method and Loyez' method for myelin sheaths are further important omissions.

Altogether it is a book which will well repay careful reading, but which, to the neuropathologist at any rate, is not likely to prove of much value as a work of reference.

Der Kokainismus. Von HANS S. MAIER. S. 269. Leipzig: G. Thieme. 1926. Price 15 M.

Among the drugs, the abuse of which has assumed considerable importance among Western nations and more particularly on the Continent than in England, cocaine has recently taken a place. Though the properties of coca have been known for long, it is only during the last forty years or so that pathological addiction to the drug has been a social problem.

Much interesting and useful information is brought together in this book. There is a long chapter on the pharmacological action of cocaine on both body and mind, and on the special symptoms of acute and chronic intoxication by the drug. These symptoms are carefully described and the differential diagnosis of cocainism is fully considered. Finally, treatment of addiction to cocaine is dealt with and methods for control of traffic in it are discussed. Though the problem is not at the moment of urgency in England, the volume must be welcomed as the only available complete treatise on the subject.

Psychological Healing: A Historical and Clinical Study. By PIERRE JANET. Translated from the French by E. and C. PAUL. Two volumes. Pp. 1265. London: George Allen and Unwin. 1925. Price 42s.

An impartial review of the development of a subject so popular as psychotherapy is to-day is valuable, may even be necessary, for by recalling the experiments of the past, the successes attained by older methods, and the hopes that proved false, we can more easily realize our present position and possibly see the lines on which progress can be most promisingly made.

Janet's name on the title-page assures that such a review will be comprehensive, analytical, and as fair as is possible in dealing with recent and contemporary hypotheses, many of which have been subjects of acute controversy. It is evident indeed that he has in his wide experience employed most of the methods which he describes. Further, he has no special thesis to maintain, and neither vaunts his own procedures nor criticizes unfairly those employed by others. Good points are found even in the methods of the mesmerists and

Christian Scientists, and features as ridiculous as any of their theses are discovered in some of the now popular creeds.

Those familiar with Janet's other books in their original tongue may expect in this the same orderly arrangement and fluent and graceful style; the translators can receive no greater praise than that these volumes have in no way suffered in their translation into English.

The first chapters contain a review of the earlier theories and practices that played a part in the development of psycho-therapeutics. First religious miracles, magical healings and animal magnetism are discussed, and there is then an interesting history of the growth of Christian Science and allied methods. The third chapter takes up "medical moralization," or the methods of appeal to the intelligence, reason, and to the moral and religious sentiments of the patients, especially that method introduced by Dubois and the "New Thought Movement."

The second part considers those methods that depend on the utilization of the patient's automatism, especially suggestion and hypnosis. There the author's personal experience is instructive; by them he achieved success in only about 7 per cent. of his neurotic patients, and mostly in suitably selected cases of hysteria in which the symptoms were not of long standing.

The third section, which is entitled "Psychological Economics," includes chapters on treatment by rest and isolation and by mental liquidation. His opinion is that rest and isolation, though useful in some cases, have been given an exaggerated value and may be even harmful in certain of the psychoneuroses and when applied to unsuitable cases. The chapter on mental liquidation contains a critical review of the Freudian doctrines and the various modifications of psycho-analysis. The author is evidently not enthusiastic on the value of the practical applications of these theories of the Vienna school and picks out for criticism, often with a playful irony, the weak points of their teaching.

The second volume begins with a review of those methods which aim at enabling the patient to acquire new tendencies and to recover those which have been lost. The first of these methods he considers is education and re-education in their application to functional motor disorders, as well as suggestions for mental gymnastics and the education of attention, will and the emotions. A more detailed description of his procedures would have been welcome, though a fair idea of them may be obtained from the case records scattered through the pages. "Treatment by excitation" follows, in which are included all those agents, as work, occupation, environment, emotional and other stimuli, which can lead to an increase of psychological tension. Psycho-physiological methods, with the aim of improving or correcting bodily health, are also referred to, though the author believes their importance in the treatment of neuroses is usually exaggerated. The final chapter is headed "moral guidance," but the title "personal influence" would better describe its contents.

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EDITOR.